



THE BRITISH  
JOURNAL OF SURGERY





# THE BRITISH JOURNAL OF SURGERY

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## FASCICULUS I: TUMOURS OF BONE.



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## EPONYMS.

By Sir D'ARCY POWER, K.B.E., LONDON.

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### XVII. CHARCOT'S JOINTS.

ALTHOUGH Charcot was a physician, it is befitting that the BRITISH JOURNAL OF SURGERY should share in commemorating one who was ever friendly to the English-speaking nations, and they in return were the first to use the term 'Charcot's joints'.

Jean Martin Charcot was born in Paris on Nov. 29, 1825. His father was a coachbuilder, who told his three sons that he could not bring them all up to a liberal profession. The one who did best at the end of his year at school should continue his studies, the second should become a soldier, and the third should be a coachbuilder like himself. Jean Martin did best, and was sent on to the Lycée Saint-Louis, where he remained until he began his medical education in 1844. He became 'Interne des Hôpitaux' in 1848, was appointed to the Salpêtrière, and collected there the material for the inaugural thesis on arthritis nodosa with which he graduated M.D. in 1853. For the next two years he served as 'Chef de Clinique', making money enough by private teaching to repay his parents for the pecuniary sacrifices entailed by his education. In 1856 he was Physician to the Central Bureau of the Paris Hospitals, and four years later he became Professor Agrégé. In 1862 he was appointed Physician to the Salpêtrière, an asylum for the aged and infirm, where he remained until his death. He succeeded his friend Vulpian as Professor of Pathological Anatomy in the medical faculty in 1872, and performed the duties until the faculty, in 1882, asked that a Chair of Clinical Neurology should be attached to the Salpêtrière, and Charcot chosen to fill it. He was elected a member of the Academy of Medicine in succession to Cruveilhier in 1872, and in 1883 he became a member of the Institute. He died of angina on Aug. 16, 1893, unexpectedly, although it had been clear to his friends that his health had been failing for some time before his death.

In person Charcot bore a strong resemblance to the Bonapartes, and more especially to Prince Jérôme. He was a man of wide interests: music, painting, art, and literature equally attracted him, and thanks to a fortunate



and happy marriage he was able to cultivate these hobbies as well as the purely scientific side of his profession.

Arthropathies, or affections of the joints associated with injuries of the nerves and diseases of the nervous system, had already attracted the attention of Weir Mitchell and Duchenne, but it was left to Chareot to study them carefully and to differentiate them from gout, rheumatism, and what is now termed osteo-arthritis. Tabetic arthropathy interested him from the beginning of his teaching at the Salpêtrière. He only recognized at first the hydrarthrosis which is of not infrequent occurrence in locomotor ataxy. The dramatic suddenness with which it appears, and its gradual improvement in many cases, led him to a closer study, and he discovered that a form existed in which



JEAN MARTIN CHARCOT.

*From an Etching by Professor Paul Eucher (1891).*

the articular ends of the bones underwent great and rapid changes. The preparation (*Fig. 1*) which he presented to the Museum of the Royal College of Surgeons in 1877 (*General Pathology*, 1022.1) was amongst the first, if not the very first, which he described in detail. He says (*Archives de Physiologie normale et pathologique*, 1869, ii, 121):—

“A woman aged 49 was admitted to the Salpêtrière on May 1, 1867. She had begun to suffer from symptoms of locomotor ataxy ten years previously, and she had been bedridden for the last four years. She noticed on awakening in the morning of June 9, 1860, that her left shoulder was swollen, the swelling getting progressively less until it reached the wrist. She was not feverish, there was no pain, and she was quite unable to account for the swelling.

Three days later the swelling had disappeared from the arm and forearm, but the shoulder still remained larger than normal, and there was marked creaking when the joint was moved.

"On June 18, in addition to the general swelling of the shoulder, there was a rounded swelling about the size of an orange situated in front of the joint. It fluctuated, and appeared to be a distended subdeltoid bursa. The creaking in the joint was still more marked. Matters remained in this state until Aug. 2, when the patient was seized with diarrhœa, choleraic in character, of which she died on Aug. 15. The swollen shoulder became much smaller a few days before death.

"A post-mortem examination made on Aug. 16 showed that the capsule of the joint was much thickened, and contained some bony plates in its substance. The synovial membrane was also thickened, and was slightly reddened on its inner surface. The cavity of the joint contained a little transparent yellow fluid, and there were intra-articular loose bodies. The head of the humerus had undergone the remarkable changes shown in the accompanying figure (Fig. 1). Although the arthropathy had only lasted nine weeks, a large part of the head of the bone had disappeared as if it had been rubbed away by friction. There was no trace of the articular cartilage, and the globular head was replaced by a flat or slightly concave surface, worn away and roughened in some parts, smooth and eburnated in others. A few small rounded osteophytes surrounded this surface, but they were quite unlike the bony edges which surround and enlarge the joint surfaces in osteo-arthritis.

"The glenoid cavity showed similar changes. The surface was worn away like the head of the humerus, but to a lesser extent. Every trace of articular cartilage had disappeared, and there was no lipping of the bone. The clavicle and acromion were normal in appearance. The posterior columns of the spinal cord showed in a high degree the lesions of grey degeneration with atrophy, more especially in the dorsal and lumbar regions. The cervical cord was affected in the same way, but to a less extent. The posterior nerve-roots were clearly atrophied and of a greyish colour. There were slight traces of a posterior spinal meningitis."

Charcot must have prized this specimen very highly, for the woodcut illustrating it is used in his *Leçons sur les Maladies du Système nerveux faites à la Salpêtrière*, Paris, 1873, ser. ii, p. 65; in the *Leçons* of Jan. 1, 1875, 2nd ed., p. 120; and in the English translation of the *Lectures on the Nervous System*, published by the New Sydenham Society in 1881, ser. ii, p. 56.

The lesions in tabetic arthropathy became common knowledge in England after the International Medical Congress held in London in 1881, when Charcot gave a remarkable demonstration of specimens. He showed the wax model of a woman, age 60, as one of the best existing specimens exhibiting the

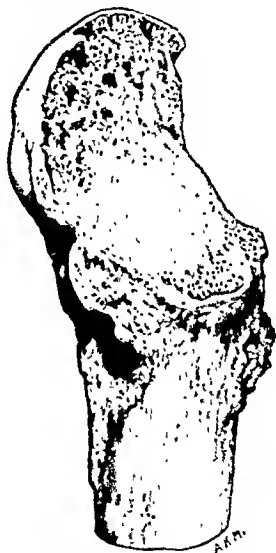
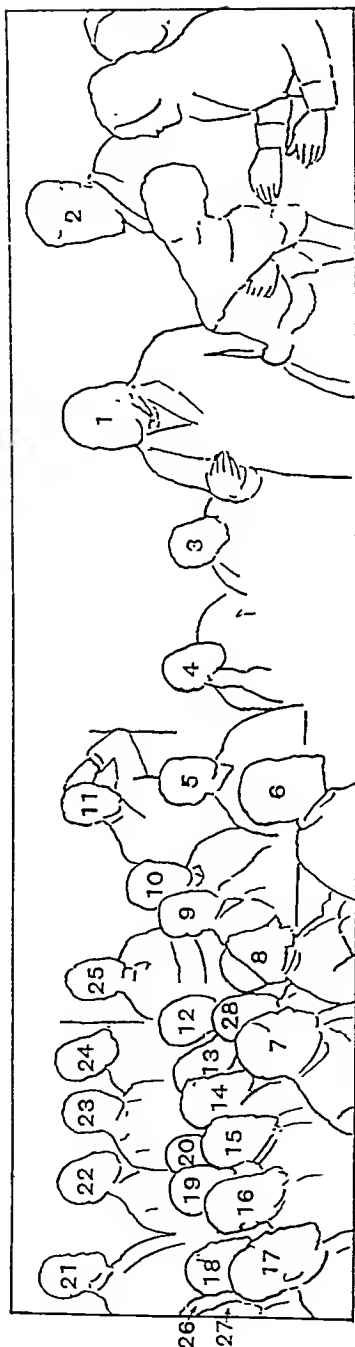


FIG. 1.—Tabetic arthropathy, showing changes in the head of the humerus.



A DEMONSTRATION BY DR. CHARCOT AT LA SALPÊTRIÈRE.

## KEY BLOCK TO THE ILLUSTRATION.



- |                              |  |                       |
|------------------------------|--|-----------------------|
| 1. Dr. Charcot               | 11. Dr. Joffroy                            | 21. Dr. Cornil        |
| 2. Dr. Babinsky              | 12. Dr. Ballet                             | 22. M. Burty          |
| 3. Dr. Paul Richer           | 13. Dr. Bourneville                        | 23. Dr. Debove        |
| 4. Dr. Féré                  | 14. Mr. Naquet                             | 24. Dr. Mathias Duval |
| 5. Dr. Marie                 | 15. Mr. Charette                           | 26. M. Jean Charcot   |
| 6. Dr. Gilles de la Tourette | 16. M. Lebas (Directeur de la Salpêtrière) | 26. Dr. Le Lorrain    |
| 7. M. Henry Berbez (interne) | 17. M. Loude                               | 27. M. Ribot          |
| 8. Dr. Vigouroux             | 18. M. Guinon                              | 28. Dr. Parinaud      |
| 9. Dr. Paul Berbez           | 19. Dr. Gombault                           |                       |
| 10. Dr. Brissaud             | 20. Mr. Paul Arène                         |                       |

*This plate, from the picture by A. Brauillet, is reproduced by the kind permission of the Longstaffe. Le François, 91, Boulevard St. Germain, Paris, the publishers of the original engraving, from whom copies (22 x 17 in.) may be obtained.*

character of ataxic affections of the joints. He afterwards presented the model to the museum of St. Thomas's Hospital, where it may still be seen under a glass case on the floor of the museum. He also gave a shoulder-joint showing tabetic arthropathy to the museum at Manchester. Sir James Paget, President of the Congress, wrote on Aug. 5, just before this remarkable exhibition :—

“ My dear Professor Chareot,

“ As you will be speaking on the morbid conditions of the joints and the bones associated with locomotor ataxy, I beg you to let me call your attention to a question, whether these are not instances of a disease which has lately for the first time appeared, or, at least, has lately become much more frequent than formerly it was.

“ There is, I think, evidence for this opinion in the fact that specimens of this disease are rarely, if ever, to be found in any but the most recently collected museums. I can speak positively of only some of those in London, but among them are the museums of the Royal College of Surgeons and of St. Bartholomew's Hospital, in which are specimens of diseased bones and joints collected from 1770 to the present time. Till the time at which you called attention to these morbid states, neither of these museums contained one specimen ; and yet they include all the examples of diseased bones collected by many surgeons and good anatomists, some of whom preserved every bone that appeared to them strange or rare. I refer particularly to John Hunter, Howship, Langstaff, and Stanley, of whom I can be quite sure that if they had ever seen specimens of the disease which you have described, they would have regarded them as treasures to be carefully preserved. No specimen has been preserved by any one of them ; and yet they gathered all that was unusual in morbid anatomy from hospitals, dissecting rooms, workhouses, and graveyards.

“ I take the liberty of suggesting this question to you as one of great interest, not only in general pathology but in the history of diseases. Besides it may lead many to believe, as I do, that a good pathological museum may be a valuable historical record.

“ Believe me, my dear Professor, sincerely yours,

JAMES PAGET ”.

A note is appended to this letter, probably by the Honorary Secretaries of the Congress, Sir William McCormac and (Sir) George Makins, that : “ It is extremely remarkable to relate, and pathologically as well as historically noticeable, that neither in the great museums of France or England, nor any place known, prior to the observation of Charcot, were there any characteristic specimens of this disease. Thus in the Musée Dupuytren, which is usually known as containing fine specimens of all sorts, and is rich in bone disease, there was no specimen showing this disease of the bones, until Professor Charcot presented three or four specimens of the kind. The disease is, in fact, a distinct pathological entity and deserves the name, by which it will be known, of ‘ Charcot's disease ’ ”. And by this name it is known to this day.

Many specimens of Charcot's joints have been collected and preserved since 1881, and the Museum of the Royal College of Surgeons of England alone contains seventeen examples of the affection.

## TUMOURS OF THE TESTICLE: THE TERATOID GROUP.

BY F. GORDON BELL, EDINBURGH and DUNEDIN.

THE material forming the basis of this investigation consists of fifty specimens of testicular tumours nearly all of which are preserved in the Edinburgh University Surgical and Anatomical Museums, the others being kindly lent by friends. These two collections appeared likely to contain an adequate representation of the commoner new growths of the testicle, and likely also to offer a fair estimate of the relative frequency of the different types. Only these cases are included in the statistical review; but, in addition, ten other specimens were selected from the Museum of the Royal College of Surgeons of Edinburgh with the object of further investigating the teratoid group, and free use is made of any points elicited from them in the general consideration of the teratoid tumours and their wider application.

Certain difficulties are attached to an investigation of testicular tumours on any scale. In the first place they are rare tumours, estimated recently by Southam and Linnell to appear once in 1500 male surgical hospital admissions, a figure which corresponds closely to Russell Howard's estimate of the London Hospital cases. Consequently, many of the specimens appearing in surgical museums are of considerable antiquity, and likely to be past their best from the histological point of view. The writer has, however, been agreeably surprised at the excellent state of preservation of many of the older specimens, which is good enough for more than the cruder recognition of the various blastodermic derivatives in teratoma, though occasionally found wanting when the interpretation of the more exact cellular pathology is at stake.

Anyone investigating a fairly large collection of testicular tumours will undoubtedly encounter many puzzles. These lie not so much in the multiple-tissued tumours—for, once a teratoid origin is suspected, further examination usually produces confirmatory evidence—but centre rather in the so-called medullary, highly cellular, apparently homogeneous variety, when it is sometimes difficult to say whether a particular specimen is predominantly cancerous or sarcomatous, and still more difficult to arrive at an estimate of its precise origin. The investigator will, however, get his reward in the education provided by the great variety of pathological tissues which come up for review.

No subject in the entire range of surgical pathology has produced more diversity of opinion than the interpretation and attempted classification of new growths of the testicle. They are regarded by some as predominantly cancerous, by others as sarcomatous, and yet again as teratoid in character,—and recent papers show that there is still little unanimity of opinion. Nicholson, in Great Britain, did good service by demonstrating that the

traditional frequency of sarcoma was much over-estimated. Chevassu, in France, brought into prominence the *séminome* or spermatocytoma—a cancer of supposedly seminal-celled origin, regarded by some as the most frequent new growth of the testis, while Wilms, in Germany, and Ewing, in America, have done much to advance the teratoid aspects of the problem.

It may be stated at once that, so far as observations on the present series go, it appears that the vast majority of testicular tumours fall into two main categories: (1) The *teratoid* group; (2) The *germinal-celled* variety, which is variously designated germinal-celled carcinoma, seminoma, spermatocytoma, embryonal carcinoma, and round-celled sarcoma. The teratoid group has predominated to a marked degree, and is discussed here, leaving the consideration of the more difficult highly cellular type to a second paper. To avoid repetition, only certain specimens are described in detail, and these are selected and grouped as far as possible to illustrate important points which have a direct bearing on the wide field of testicular neoplasms in general.

Though no vital new facts emerge from this investigation, it is hoped that the following presentation of the subject may help to clarify some of the difficulties attached to it. I am greatly indebted to the publishers for their generosity in the matter of microphotographs, which go far to illustrate the main points.

### TERATOMA TESTIS.

#### General Considerations.—

DEFINITION.—‘Teratoma’ testis may be defined as a tumour composed of elements derived from all *three* layers of the blastodermic vesicle, the derivatives being represented in differing degrees and in varying grades of complexity. This definition is too rigid, for, as will be shown presently, there are many cases in which representative elements of only *two* layers of the blastoderm are present, and, proceeding further, there is every probability that frequently only *one* layer is represented.

The term ‘teratoma’ should perhaps be reserved for the rare, highly complex types displaying structures which can reasonably stand interpretation as a successful attempt on the part of the tumour to produce adult or fully formed structures or organs such as the alimentary tract, bones, kidney, thyroid, breast, spinal cord, and so on; while the term ‘teratoid’ should be applied to the common, less complex types where the activities of the tumour are abortive and produce only a mixture of tissues. As numerous gradations occur and indicate that these tumours all belong to the same family, the terms ‘teratomatous’ and ‘teratoid’ are used synonymously in the following sections. The term ‘mixed tumour’ often applied to the teratoid group is in this article restricted to a special type which resembles certain parotid tumours.

There is a tendency on the part of one school of pathologists to extend the scope of teratoma testis, and to regard almost all testicular tumours as of teratoid origin, with the highly cellular homogeneous varieties classed variously as sarcoma and carcinoma by other observers as one-sided developments of the teratoma. This conception, the germ of which dates back to the pioneer work of Koehler and Langhans,<sup>1</sup> has the able advocacy of Ewing,<sup>2</sup> and is highly attractive, as providing a common origin for the great variety of tumours met

with in the testis; but in the writer's opinion it goes too far, and the evidence against this universal application will be discussed in a second paper.

**TERMINOLOGY.**—It is convenient here to refer to the older terminology applied to testicular tumours. The teratoid group has in the past paraded under various designations, of which perhaps the most popular has been 'fibrocystic disease', a term receiving justification from the naked-eye and cruder microscopic appearance of a cyst supported by a matrix of fibrous tissue. Various other terms, such as 'chondroma', 'chondrosarcoma', 'chondro-adenoma', 'osteoma', 'adenoma', 'squamous- and columnar-celled carcinoma', are referable to the most obvious histological features occasionally displayed which have dominated the mind of the pathologist. These older terms serve as an indication of the remarkable structural variations presented by these tumours, which are now regarded as variations from a common teratoid basis.

**Macroscopic Features.**—Teratoma testis presents variations in size from a golf ball up to the foetal head. The surface may be smooth or lobulated. may appear uniform in consistence, or may be soft or even fluctuant in parts, and sometimes stony hard. The internal structure is best demonstrated by (vertical) sagittal section, which brings out the relation of the tumour to the body of the testis and to the epididymis, and may in early specimens clearly demonstrate its frequent origin from the mediastinum testis. In the early stages the body of the testis is usually visible to the naked eye at some part of the periphery of the tumour, commonly at the summit, and may be fairly normal in shape and size, while in large tumours it undergoes progressive compression till it is reduced to a narrow strap-like peripheral layer only detectable under the microscope. The presence of a peripheral band of testicular tissue separated from the tumour substance by a condensed fibrous layer or capsule is highly characteristic of teratoma, but not pathognomonic (*Fig. 2*).

The cystic character is often evident, the cysts varying in size from a pin's head to a marble, or larger. In some of the older specimens the appearance is most aptly compared to a coarse sponge, while in recent cases the cut surface is often honeycombed and is strongly reminiscent of a colloid goitre, and the cyst contents are serous, mucoid, gelatinous, or blood-stained. The cystic character may be general or patchy, and considerable parts of the cut surface may present a solid homogeneous appearance. As in other cystic



**FIG. 2.**—*Teratoma*: Low-power view showing general structure. Remains of testis at periphery, separated from the cystic teratomatous tissue by a capsule or condensation of fibrous tissue. Numerous spaces of an intestinal character (hypoblast), one at left edge containing a rod of stratified epithelium capped by horny laminae (epiblast), and two small pale nodules of cartilage (mesoblast).



tumours, irregular cavities may result from breaking-down of the cyst walls. While a cystic appearance is highly characteristic of teratoma testis, numerous apparently solid tumours occur in which the teratoid character comes as a surprise on microscopic examination.

Cartilaginous nodules are sometimes visible in the cut surface, and in rare cases may appear to the naked eye to form the bulk of the tumour, and to justify the old term of 'chondroma testis'. Like the cysts, the cartilaginous islets vary greatly in size and distribution, some being only just visible, while others appear as nodules the size of a cherry.

### Microscopic Features.—

**METHOD OF STUDY AND GENERAL REMARKS.**—The histological characters and general structure of testicular tumours are undoubtedly best studied by means of the 'whole section' method. This, unfortunately, has been only partially available, and it has been necessary to fall back on the more laborious and less satisfactory plan of taking individual sections from various parts of

the tumours. It is important to pay attention to the periphery, and especially to the region of the upper pole of the specimen, for it is here that the remains of the testis proper are so often encountered, and the discovery of testicular tissue separated from the neoplastic tissue by a condensation of fibrous tissue is strongly presumptive evidence of a teratoid origin. It cannot be emphasized too strongly that the examination of a small piece of these complex tumours may be highly misleading. Again and again repeated sections have demonstrated the teratoid character in cases where the first section suggested a simple cellular tumour, and furthermore the various blastodermic derivatives show an occasional tendency to be aggregated in one area and appear to dominate the histological picture in the absence of further examination. Thus, in *Specimen 1*, presently described, the striking

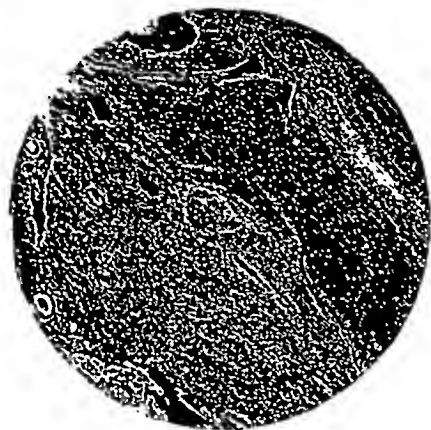


FIG. 3.—*Teratoma*: Showing general structure and representative elements of all three layers of the blastoderm in the same field. *Mesoblast*: Nodule of darkly-stained cartilage near centre, and abundant fibro-myxomatous matrix. *Hypoblast*: Various spaces lined by columnar or cubical epithelium. *Epiblast*: Rod of stratified epithelium bounded by Malpighian layer (right).

feature in the original section taken fifteen years ago was the presence of stratified epithelium with typical cell-nest formation, which naturally suggested a diagnosis of epithelioma; while in recent sections hypoblastic elements of an intestinal character markedly predominated. Similarly, a cartilaginous formation may appear to be the chief or even sole feature, but further search almost always reveals other elements. Paget's<sup>3</sup> much quoted malignant enchondroma is a case in point: it was finally shown by repeated investigation to contain all three blastodermic elements, and placed conclusively in the teratoid category.

In general it may be said that teratoma testis displays histological characters of a highly variable complexity, and the individual representation of the blastodermic layers varies greatly in different specimens. After examining a considerable number of these tumours it is possible to reduce them to a group type which, in order to give a composite histological picture (*Fig. 3*), may be briefly described as follows:—

1. *Mesoblastic Derivatives*.—The presence of cartilage in the form of round or oval nodules or crescentic plates is characteristic. A fibrocellular myxomatous matrix, sometimes sarcomatous in type, is almost equally characteristic, and always suggests that any tumour under consideration is teratoid in character.

2. *Hypoblastic Derivatives*.—These take the form of tubules or spaces lined by cubical or columnar cells, often suggesting adult glandular structures, especially intestinal mucosa.

3. *Epiblastic Derivatives*.—These take the form of spaces lined by stratified squamous epithelium, and cell-nest formation is often a striking feature.

To summarize, the common mesoblastic element (other than stroma) is represented by cartilage, the hypoblastic by glandular tubules or spaces, and the epiblastic by stratified epithelium. These and other derivatives will now be considered in greater detail.

**Blastodermic Derivatives Considered more Fully.**—No attempt has been made to identify all the tissues which may appear in these tumours. This is already a well-tilled field, and as there is no reason why any or all tissues and organs of the body should not be represented, the subject loses its interest. A short account of the tissues commonly met with as depicted by the present group is given with the object of considering the possible relations of these tissues to the development of tumours by a process of individual overgrowth.

1. **MESOBLAST.**—The formed elements are represented by hyaline cartilage, bone, plain and striped muscle, fat, and lymphoid tissue.

*Hyaline cartilage* is the most frequent and striking derivative, and takes the form usually of round or ovoid nodules of greatly varying size; but sometimes it appears as plates or crescents disposed around glandular spaces, perhaps suggesting the structure of the trachea. It is sometimes so abundant as to give the tumour the naked-eye appearance of an ordinary chondroma, but may be extremely scanty. It ranges in structure from an embryonic character up to the ordinary adult type, and in some of the larger nodules active proliferation may suggest a malignant change, but the nodules practically always appear to be well encapsuled by fibrous tissue or occasionally by plain muscle. Myxomatous degeneration is often a marked feature, and central cystic degeneration in the larger islets may be visible to the naked eye; but after comparison with a considerable number of mixed parotid tumours, the cartilaginous element of the teratoma testis appears to possess a better developed structure and is not so subject to gross myxomatous degeneration.

*Bone* is not a common feature, but may appear in pure form or as a partial ossification of cartilaginous nodules. The presence of ossification on an extensive scale no doubt accounts for the cases of osteoma recorded in the past.

*Plain muscle* is a common element, and may be distributed irregularly,

but sometimes surrounds cartilaginous nodules, and is frequently disposed around glandular spaces, where it may be taken to represent an attempt to form the muscle coat of rudimentary intestine. Its overgrowth may be related to the development of the rare myoma testis.

*Striped muscle* has been a rare element in the specimens under consideration, but has appeared in both embryonic and adult forms. Similarly its overgrowth may be related to the extremely rare rhabdomyoma or rhabdomyosarcoma testis.

*Heart muscle* is said to be excessively rare, and has not been observed in the present cases.

*Lymphoid tissue* is frequently present. It may have an irregular nodular distribution, but is commonly seen in association with glandular spaces lying between the epithelial and muscular layers, and doubtless representing the lymphoid tissue of the digestive tract. It is also commonly observed in malignant cellular tumours, forming a lymphoid stroma or network around the alveolar groups. It has been suggested that overgrowth of the lymphoid tissue of a teratoma may give origin to lymphosarcoma testis. This subject is discussed later, but in the meantime the frequent presence of lymphoid structures in teratoid tumours should be noted.

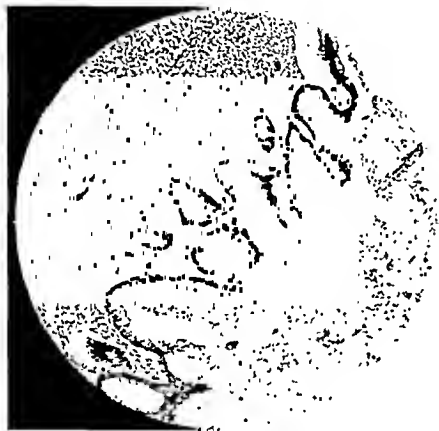


FIG. 4.—*Teratoma*: 'Intestinal tube'. Wall of space lined by columnar cells, many of which are of the goblet variety. Note the lymphoid tissue between the mucosa and the plain muscle coat.

2. **HYPOBLAST.**—This is represented by glandular tubules or spaces lined by cubical or columnar cells. Such cells may be of an undifferentiated type, representing, as it were, a rudimentary hypoblastic element as seen in the early embryo; but frequently the spaces are so arranged as to suggest that they indicate an abortive attempt on the part of the teratoma to produce typical fully-formed tissues or organs, especially digestive or respiratory tracts.

*Digestive tubes* are lined by columnar cells, and the goblet or mucigenous

type is the striking feature. Both small and large intestine may be represented, and the resemblance to adult gut is strengthened by collections of lymphoid tissue beneath the epithelium and by encircling bands of plain muscle sometimes disposed in two layers (*Fig. 4*). It is not uncommon to see tubes lined partly by glandular epithelium and partly by stratified epithelium, and such an appearance has been fancifully interpreted as representing the gastro-oesophageal junction.

*Respiratory tubes* are lined by columnar cells, sometimes ciliated, and the resemblance to trachea or bronchi is enhanced by supporting nodules or plates of hyaline cartilage, and by encircling bands of plain muscle (*Fig. 5*).

Certain glandular organs, such as salivary glands, prostate, heart, liver,

and pancreas have all been described, but have not figured in sections from the specimens under consideration, though it is not uncommon to find acini lined by cubical or columnar cells grouped together in a gland-like fashion. *Thyroid tissue* was present in three specimens, reproducing adult and foetal types, and in another tumour some adenomatous tissue presented a close resemblance to a cystic goitre.

Some of these glandular structures may be epiblastic in origin, e.g., mammary tissue; but probably the majority are hypoblastic, and it is convenient to group them together, though perhaps not always accurate.

### 3. EPIBLAST.—

*Stratified epithelium* is the most frequent and striking representative, but is sometimes scanty and hard to find, and may be absent. It assumes varying forms, usually lining cysts, but sometimes forming solid columns or plaques (Fig. 6). Typical 'cell-nest' formation is often a striking feature, and in rare cases the keratinized epithelium takes the peculiar form of circular laminæ, aptly compared with the skins of an onion. Cell-nest or epithelial pearl formation, though highly typical of squamous epithelioma elsewhere, appears to be a natural event in the epidermoid cysts of teratoma testis. Reference has already been made



FIG. 5. — *Teratoma* (same specimen): 'Respiratory tube'. Narrow space lined by columnar cells supported by plate of embryonic cartilage. This particular tube was of some length, and had several nodules of cartilage in close relation to it, cartilage being noticeably absent in other parts of the section.



FIG. 6.—*Teratoma* (same specimen): Epiblastic elements. (Left) Rod of stratified epithelium, capped by horny layers, lying in a space lined by columnar cells. (Right) Wall of epidermoid cyst, showing cell nests.

to the existence of cysts lined partly by stratified and partly by cubical or columnar epithelium, and one is inclined to regard this as indicating a partial stratification of the Malpighian layer.

*Neuro-epithelium* has been observed in several cases, sometimes reproducing the primitive structure of the spinal cord or brain, and scanty groups of ganglion cells have appeared rarely.

*Trophoblastic epithelium* has not figured in the present group.

*Melanotic patches* have occurred in several specimens, but this is uncertain ground, and an epiblastic origin cannot be positively assumed.

**Teratoma Testis and Malignant Conversion.**—It is generally stated that teratoma testis is usually innocent, but may become malignant in virtue of any of its compound elements, and that the resultant tumour may be cancerous or sarcomatous, depending on whether the malignant change affects the connective tissue or epithelial structures. Such a generalization is certainly true, but in the opinion of the writer requires qualification. The testicular teratoma so frequently displays unequivocal signs of malignant change that its essentially malignant character should be more emphatically stressed; in the second place the marked tendency of the epithelial elements, especially the hypoblastic, to assume malignant characters requires greater emphasis.

The *hypoblastic elements*, as observed in the present group, play a more dominant part than either the connective-tissue or squamous epithelial elements in conferring malignant potentialities on these complex tumours, and frequently transform them into carcinomata of different types. Many predominantly carcinomatous tumours of the testis find their precise counterpart in areas of malignant proliferation affecting the glandular structures of teratomata, and if we accept the principle that the overgrowth of the malignant element may lead to partial or complete suppression of the other blastodermic derivatives, the relationship of the teratoma to the pure or almost pure carcinoma of the testis becomes not a hypothesis but a certainty.

No doubt the *connective-tissue elements* occasionally take on a sarcomatous or endotheliomatous structure and sometimes produce purely sarcomatous metastases, and, similarly, the embryonic or adult *epiblastic elements* may assume carcinomatous characters and produce characteristic tumours; but these transformations have not figured with anything like the same frequency in the present series as malignant conversion of the hypoblastic glandular structures.

#### THE VARIATIONS OF TERATOMA TESTIS.

The specimens described below have been selected with the special object of demonstrating the principle relative to teratoid tumours of the testis that one blastodermic element may overgrow the others, thus producing tumours composed chiefly or entirely of one type of tissue, which may be innocent or malignant. The application of this principle is of far-reaching importance in the interpretation of the great variety of new growths of the testicle, and undoubtedly provides the key to a large part of this field of testicular pathology.

It is proposed to describe: (1) The typical teratoma which can be recognized at once by a glance at the cut surface, and contains representatives of all three layers of the blastoderm; (2) A solid, seemingly homogeneous tumour containing heterologous elements, in order to show that teratoid tumours frequently lack the characteristic 'cystic' appearance; and (3) Specimens illustrating individual variations which can reasonably stand interpretation as one-sided developments of a teratoma. By great good fortune many variations have occurred in the material available and provide a fairly complete series. Though several gaps exist, it does not appear to impose too great a strain on scientific credulity to assume the possible existence of the missing members. A series of sixty specimens is too small to include all types, but reference to the literature readily provides the necessary examples.

The specimens here described fall naturally into the following categories:—

1. *The typical tri-dermal teratoma*.—‘Fibro-cystic disease’ of the older terminology.

2. *The solid, apparently homogeneous tumour containing heterologous elements*.

3. *Mesoblastic overgrowth*.—(a) Cartilaginous; (b) Myomatous; (c) ‘Mixed’ tumours of myxo-chondro-endotheliomatous type resembling certain parotid tumours.

4. *Hypoblastic overgrowth*.\*—(a) Simple papillary adenofibroma with heterologous elements; (b) Adenocarcinoma with heterologous elements; (c) Pure adenocarcinoma; (d) Spheroidal-celled scirrhus carcinoma.

5. *Epiblastic overgrowth*.—(a) Dermoid cysts—so-called; (b) Squamous carcinoma (epithelioma); (c) Basal-celled carcinoma (rodent cancer); (d) Chorion-epithelioma; (e) Neuro-epithelial tumours.

Sub-groups (b), (d), and (e) of the fifth category do not occur in the present series, but are mentioned to round off the subject.

*Specimen 1*.—The typical three-layered teratoma.

The tumour was removed from a man, age 26, who had complained of pain in the testis dating from an alleged strain fourteen weeks previously.

**MACROSCOPIC**.—The specimen is bisected sagittally, the tumour and fused testis together forming a spherical mass a little larger than a golf ball. The epididymis is not involved and appears unduly mobile. The cut surface displays the cystic character of the teratoid tissue to great advantage, and is honeycombed with small pea-like rounded or oval cysts. The body of the testis is readily made out at the upper pole of the tumour, and is separated from the ‘cystic tissue’ by a definite capsule. The testis is compressed and reduced to the size of a bean, but the cut surface presents the normal characteristic pale granular appearance, and stands out in marked contrast to the cystic teratoid tissue below (*Fig. 7*).

**MICROSCOPIC**.—The teratoid nature of the tumour is at once evident, and all three blastodermic derivatives are represented, but in varying proportions.

*Hypoblastic Elements* dominate the picture, and show an advanced degree of development. They appear in the form of glandular spaces lined by columnar cells, many of them being of the goblet or mucigenous variety like those of adult intestinal mucosa. The resemblance to intestine is enhanced by subepithelial aggregations of lymphoid tissue and by encircling bands of plain muscle fibres. Other spaces lined by columnar cells supported by occasional plaques of embryonic hyaline cartilage might stand interpretation as respiratory tubes (*see Fig. 5*).

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\* It is possible that some tumours included in this group are of glandular epiblastic origin, but it is convenient to group them together.

*Mesoblastic Elements* are represented by the fibromyxomatous matrix, by plain muscle bundles, by lymphoid aggregations, and by scanty nodules of embryonic cartilage. In the main the formed mesoblastic elements are decidedly scanty.

*Epiblastic Elements* are represented by spaces lined by stratified epithelium, and perfect cell-nest formation is a striking feature (see Fig. 6). The first section taken from one part of the periphery of the tumour showed a marked preponderance of epidermoid cysts and suggested a diagnosis of squamous epithelioma, while later sections from the rest of the tumour displayed great overgrowth of the hypoblastic elements as described above.

*The Testis Proper* is disposed chiefly at the upper pole as seen by the naked eye, but a thin film of testicular substance is spread over the greater part of the free surface of the teratoid tissue and is separated from it by a definite fibrous capsule. The arrangement and shape of the seminiferous tubules appear to be normal, and spermatogenesis is evident.



FIG. 7.—*Teratoma testis*: 'Typical fibrocystic disease'. The cut surface is so characteristic that the nature of the tumour is evident at a glance, in contrast to the succeeding figures. The body of the testis is situated at the upper pole of the teratoma, and the epididymis in this case is unusually mobile.

*Comment.*—The specimen is a perfect example of the typical teratoma testis, and the appearances presented by the cut surface are so characteristic that the diagnosis is evident at a glance. The relationship of the teratoid tissue to the body of the testis and to the epididymis at once suggests that the new growth originated in the mediastinum. The tumour is small, coming to light at a relatively early stage of its development, probably owing to the patient's attention being drawn to the enlargement of the testicle by the injury. No evidence of malignant degeneration of the various blastodermic elements appears in the sections

examined, unless the epidermoid cysts are regarded as such; but it would appear that cell-nest formation is to be regarded as a normal development from cysts lined by stratified squamous epithelium. The hypoblastic tubes and spaces present such an unmistakable resemblance to adult intestine that the tumour merits a position in the teratoma category rather than the teratoid.

This highly characteristic three-layered tumour should be regarded as the fundamental type and the basis of the variations described below.

*Specimen 2.*—Solid, apparently homogeneous tumour with heterologous elements.

No history is attached to this specimen, which belongs to the Spence collection.

**MACROSCOPIC.**—The tumour is the size and shape of a grape-fruit and is bisected sagittally. The cut surface presents all the appearances of a solid homogeneous structure. That the tumour was not homogeneous was first indicated by a gritting sensation on taking pieces for microscopic examination.

**MICROSCOPIC.**—*Hypoblastic Elements.*—The bulk of the tumour is formed of masses of spheroidal or oat-shaped cells disposed diffusely or in an alveolar fashion and enclosed by septa of fibrous tissue. This malignant tissue stains poorly and for the most part gives little indication of its origin, but in a few of the spaces it is possible to make out a peripheral lining of cubical cells which merge into the oat-shape and suggest an origin from hypoblastic elements. This impression was strengthened by finding in later sections spaces lined by cubical and columnar cells.

*Mesoblastic Elements.*—The striking feature appearing in most sections is the presence of nodules and plates of well-formed bone which appear to be scattered throughout the greater part of the tumour (Fig. 8). The arrangement of some of the osteoid tissue gave the impression at first that it might possibly have developed in fibrous septa as the result of metaplasia, but discovery of patches of voluntary muscle and fatty tissue strengthened a teratoid origin.

*Epiblastic Elements* are not recognizable in any of the sections.

The *Testis* appears as a peripheral band on the surface of the tumour.



FIG. 8.—*Teratoma* : Showing osteoid tissue.



FIG. 9.—*Teratoma testis* : Mesoblastic (chondromatous) overgrowth. Cartilage is particularly evident at the lower pole in the photograph, though to the naked eye it is abundant throughout the cut surface.

**Comment.**—The specimen clearly belongs to the teratoid group, and is interesting as showing the complex nature of an apparently solid homogeneous tumour. Some of the sections showed only the malignant cellular tissue, and in the absence of further examination and the clue afforded by the gritting observed in taking the first piece for histological examination, the teratoid nature of the specimen might have escaped notice. It is a commonplace in the literature of testicular tumours to find that re-examination reveals the presence of heterologous elements in cases hitherto regarded as purely sarcomatous or carcinomatous—a sure indication of the futility of depending on the examination of a small piece from one part.

**Specimen 3.**—*Mesoblastic overgrowth*: (a) Cartilaginous.

No history is attached to this specimen, which belongs to the Spence collection.

**MACROSCOPIC.**—Only one half is preserved, and indicates that the tumour was the size of a small melon. The cut surface presents a striking resemblance to that of an ordinary



chondroma, and is studded with pearly cartilaginous nodules ranging in size from a small bead to a bean or cherry. Several of the larger nodules show central cystic degeneration. Towards the lower pole the surface appears entirely cartilaginous (*Fig. 9*), but elsewhere the cartilaginous islets are supported by strands of fibrous tissue which is nowhere abundant except near the upper pole. Several small isolated cysts are present. The remains of the testis are not visible to the naked eye.

**MICROSCOPIC.**—*Mesoblastic Elements.*—The striking feature in sections taken from different areas is the presence of numerous nodules of well-developed hyaline cartilage. These constitute the greater bulk of the tumour, and are surrounded and separated from each other by a small amount of fibrous tissue and occasionally by plain muscle-fibres. Myxomatous degeneration is evident in some of the nodules, but the cartilage cells appear to be circumscribed and do not invade the fibrous tissue. In addition to the obvious well-developed cartilaginous islets there are in some areas enormous numbers of tiny nodules containing only a few cartilage cells (*Fig. 10*).

Further evidence of the predominance of the mesoblastic element in this tumour is provided by scattered cylindrical cross-striped muscle fibres and globular sarco-blasts, while towards the upper pole there is a considerable amount of fibrous tissue.



FIG. 10.—*Teratoma*: Cartilaginous overgrowth. (Left) Showing nodules of cartilage, large and small, which make up the bulk of this tumour. (Right) Showing patch of striped muscle.

*Hypoblastic and Epiblastic Elements.*—At first sight it appeared likely that these elements had been entirely suppressed, for though scanty cystic spaces were observed which by analogy with appearances seen in other teratoid tumours might be taken to represent glandular spaces, conclusive evidence of the existence of other blastodermic derivatives was obtained only by the discovery of one large space lined by columnar cells, assuming a pavement form at one part.

The *Testis* is present at the periphery of the tumour in the form of a few compressed and atrophied tubules.

*Comment.*—The specimen is a teratoma and is a fine example of the 'chondroma testis' of the older terminology. It illustrates a remarkable overgrowth of the mesoblastic element, hyaline cartilage predominating. The importance of this type of tumour lies in the fact that it establishes the principle of the overgrowth in teratoid tumours of one blastodermic derivative at the

expense of the others—a principle which beyond doubt has a wide application in the interpretation of testicular new growths.

It is a matter of great importance in this connection to demonstrate the existence of other blastodermic derivatives in tumours composed almost entirely of mesoblast, to exclude an origin by a process of metaplasia from the mesoblastic tissue constituting the fibrovascular matrix of the testis proper. Virchow in particular allotted great scope to the influence of metaplasia in the production of complex tumours of the testis, but a teratomatous origin appears certain if the presence of characteristic elements derived from one or both the other blastodermic layers is established.

Paget's classical case of enchondroma testis, described in 1855, throws an interesting light on the phases of interpretation through which a complex tumour may pass. It was regarded originally by Paget as a malignant cartilaginous tumour producing metastasis in the lung by invasion of the spermatie veins, but later investigation by Kanthack and Pigg demonstrated the presence of columnar-celled elements, and the tumour was then looked upon as a columnar-celled cancer with cartilaginous formation in the stroma. Further investigation by Nicholson showed the existence of epithelial pearls in both the primary and secondary growths, thus establishing the existence of representatives of all three layers of the blastoderm and conclusively proving the teratoid nature. The pulmonary growth may be regarded in the light of a transplantation through the venous system rather than an evidence of malignancy.

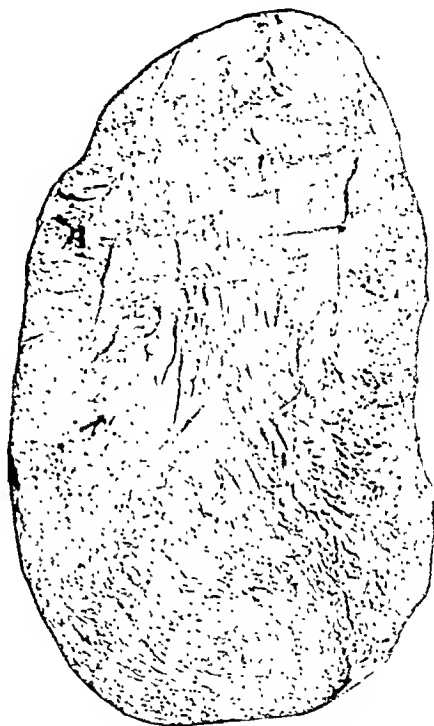
*Specimen 4.*—**Mesoblastic overgrowth:**  
(b) **Myomatous.**

No history is attached to this specimen, which belongs to the Spence collection, and the one half preserved suggests that the complete tumour was the size of a small melon.

**MACROSCOPIC.**—The cut surface gives some indication of the predominating myomatous nature of the tumour, and is fleshy and firm, and pale pink in colour over the greater part, with here and there granular areas suggesting a glandular structure (*Fig. 11*).

**MICROSCOPIC.**—*Mesoblastic Elements.*—Numerous sections taken from various parts present the same structure. The tumour is formed almost entirely of a combination of plain and striped muscle fibres, disposed diffusely or in alveoli. Sections stained by van Gieson's method display an alveolar arrangement, the muscle fibres and cells being supported by a moderate amount of fibrous tissue.

The plain muscle calls for no comment, and its arrangement recalls that of



*FIG. 11.*—*Teratoma testis*: Mesoblastic (myomatous) overgrowth. The cut surface gives some indication of the composition of this tumour.

myoma uteri. The striped muscle appears in various stages of development, and it is noteworthy that typical adult cylindrical fibres are not present in the sections examined. The developing fibres or sarcoblasts appear as pear-shaped or globular

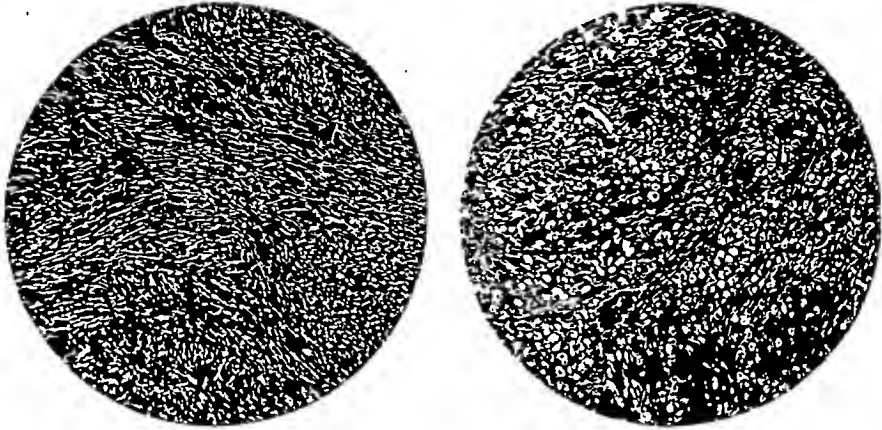


FIG. 12.—*Teratoma*: Myomatous overgrowth. (Left) Resembling ordinary myoma, but some of the spindle cells are striated, and the scanty, larger, dark, fusiform cells are developing striped fibres. (Right) Showing large numbers of globular sarcoblasts, some of which are becoming pyriform.

cells with large nuclei. Some of the sarcoblasts are transversely striated and show transition into thick fusiform striated cells (Figs. 12 and 13). Certain elongated spindle-cells closely resembling plain muscle-fibres occur here and there and show striation, and the appearance suggests that plain fibres may become striated. This point has been noted by other observers.

*Hypoblastic and Epiblastic Elements.*—

None can be distinguished with certainty, but clear spaces with a definite outline occur in various parts, and, as the specimen is old, may represent glandular spaces out of which the epithelium has been shed; consequently one would be unwilling to affirm the absence of other than mesoblastic elements in the tumour.

*Testis.*—No trace of the testicle appears in the various sections.

*Comment.*—The specimen, so far as the examination of the available half is concerned, demonstrates a remarkable overgrowth of the muscle element and merits the name rhabdomyoma or rhabdo-leio-myoma. It seems highly probable that it has arisen from an overgrowth of the



FIG. 13.—High-power view of Fig. 12, showing the characters of the sarcoblasts.

mesoblastic elements of a teratoma, and though this cannot be positively assumed, analogies presented by other one-sided developments of teratoma

testis give reasonable justification for including it in the teratoid category. Further support to the teratoid origin is afforded by the fact that other observers have demonstrated epithelial and cartilaginous structures in tumours of this type. Rokitsky's classical case of a rhabdomyoma attached to the lower pole of the testis is generally regarded, on the other hand, as a tumour derived from the museular tissue of the gubernaculum.

*Specimen 5.—Mesoblastic overgrowth: (c) The 'mixed' tumour.*

For the purpose of this paper this term is restricted to the rare tumours of the myxo-chondro-endotheliomatous type presenting a close resemblance to certain tumours of the parotid, though it should be stated that salivary-gland tumours of this variety are regarded by some pathologists as myxo-chondro-carcinoma and not as endothelioma. Two occur in the present series and merit a brief description. Both are old specimens in a poor state of preservation, but the sections are sufficiently good to display the main structural points. *Fibrous and myxomatous tissue* predominate, accounting for the poor staining. The *endothelium* of the vascular spaces is unduly prominent and shows patchy proliferation. In some areas the swelling of the endothelial cells produces a spurious adenomatous appearance, in others the cells are arranged in the form of anastomosing cords, and in others again the endothelial proliferation is so diffuse as to produce solid cellular sheets. Areas of degenerating myxomatous cartilage are present in the one tumour, but the other shows merely a *myxo-fibro-endotheliomatous structure* (Fig. 14). Both resemble degenerating salivary tumours in the disappointing quality of the sections.



FIG. 14.—*Teratoma: Myxo-chondro-endothelioma* type resembling certain parotid tumours. Interlacing framework of darkly staining endothelial (?) cells surrounding and merging into myxomatous areas, in some of which degenerated cartilage is present.

*Comment.*—Tumours of this simple type apparently composed of purely mesoblastic tissues may originate from:—

1. An overgrowth of the mesoblastic elements of a teratoma.
2. An overgrowth of the fibrovascular stroma of the testis proper with cartilaginous metaplasia.

From a consideration of the present specimens the writer inclines to the teratoid origin, because the remains of the testis can be seen at the periphery of one of the tumours, but more especially because a similar structure is often seen in patchy distribution in undoubted teratomata. From the practical point of view they can be regarded as among the most innocent of testicular tumours, and do not recur after castration. Parotid tumours, on the other hand, treated by enucleation, may recur from outlying portions left *in situ*.

*Specimen 6.—Hypoblastic overgrowth: (a) Simple papillary adenofibroma with mixed elements.*

No history is attached to this tumour, which is preserved in the Anatomical Museum, and is described as probably sarcomatous.

**MACROSCOPIC.**—The specimen is the size of a grape-fruit, and has been partially

split in the sagittal direction and spread out in front of the cord and epididymis. The body of the testis is indistinguishable. The cut surface is homogeneous and is divided into a number of large lobules by deep branching furrows, the whole appearance recalling the cut surface of certain fibro-adenomata of the breast, or of certain myxomata (Fig. 15).

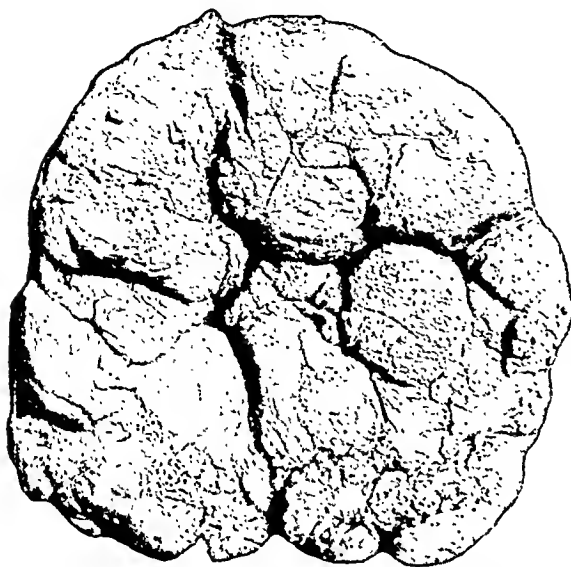


FIG. 15.—*Teratoma testis*: Adeno-myxo-fibroma. The lobulated cut surface recalls certain adenomata of the breast.

**Microscopic.**—The bulk of the tumour is composed of adenofibromatous tissue which resembles nothing so much as a mammary proliferous or papillary fibro-adenoma (Fig. 16). The papillary arrangement is readily made out, though many of the slender processes appear in cross-section. The processes are covered by flattened or cubical epithelium supported by a fibromyxomatous core, the fibrous tissue being scanty in the main though abundant here and there. No evidence of malignancy appears in any of the sections.

Sections from the periphery show mixed elements in the shape of islets of cartilage and myxomatous areas. No trace of the testis occurs in sections examined.

**Comment.**—This tumour illustrates a rare type which is certainly best interpreted as a variation of teratoma testis, the predominant adenomatous tissue representing a simple overgrowth of the glandular elements, while the mixed elements at the periphery composed of mesoblastic derivatives confirm the essential teratoid origin. The precise nature of the glandular element which by its overgrowth has produced the bulk of the tumour remains uncertain, but the resemblance to the proliferous mammary tumour is suggestive, and it is interesting to recall that adenofibromatous tumours of the breast in rare cases contain mixed elements. On the other hand, any adenomatous tissue may develop papillary projections. Papillary adenomatous tumours of the testis have been recorded very rarely in the literature, and the above example came as a surprise, though perhaps the peculiar lobulation of the cut surface should have afforded a clue.



FIG. 16.—*Teratoma*: Papillary myxo-adenofibroma, resembling certain mammary tumours.

*Specimen 7.*—Hypoblastic overgrowth: (*b*) Adenocarcinoma with heterologous elements.

The specimen was removed from a patient, age 26, who had complained of pain and swelling of the testicle for ten weeks.

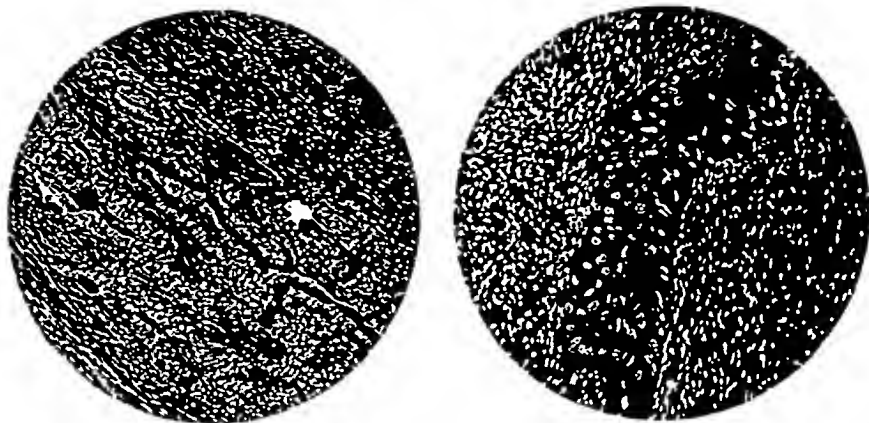


FIG. 17.—*Teratoma*: (Left) Adenocarcinoma, (Right) Space lined by stratified epithelium surrounded by abundant myxofibromatous stroma.

**MACROSCOPIC.**—The tumour is the size of an orange and is bisected. The cut surface appears homogeneous in the main, but here and there cystic formation occurs on a small scale, but is not sufficiently evident to suggest 'cystic disease'. The epididymis is not involved and is spread over the posterior surface.

**MICROSCOPIC.**—The specimen is remarkable for the histological variations presented, but in the main is an adenocarcinoma supported by a fibromyxomatous matrix.

**Hypoblastic Elements.**—Numerous tubules and spaces lined by cubical and columnar cells predominate. Some are clearly innocent, but the majority present the features of an active adenocarcinoma (Fig. 17). In some areas the infiltrating glandular cells assume a spindle shape, and being massed together give an appearance like a spindle-celled sarcoma. In several areas the cystic spaces are dilated and lined by flattened or cubical cells, and the appearances are strongly reminiscent of a simple cystic goitre. In other areas the glandular tissue assumes an infiltrating malignant papillary formation, and cross-sections of papillæ yield a spurious peritheliomatous appearance. This malignant papillary



FIG. 18.—*Teratoma* (same tumour): Papillary adenocarcinoma. This should be compared with Fig. 16, of which it appears to be the malignant version.

tissue is most aptly compared with the features seen in some malignant papillomatous ovarian tumours (Fig. 18).

**Epiblastic Elements.**—These are decidedly scanty, but appear in the form of

spaces lined by stratified epithelium and of scattered patches of pavement epithelium. No cell nests are detectable.

*Mesoblastic Elements.*—There is an abundant fibromyxomatous matrix which in some areas appears sarcomatous, though a sarcomatous appearance is certainly produced by the infiltrating glandular cells taking on a spindle shape. Myxo-endotheliomatous areas are occasionally abundant. No cartilage or other formed mesoblastic elements appear in any of the sections.

*The Testis* is readily recognizable as a flattened band at the surface of the tumour.

*Comment.*—The specimen is a teratoma, hypoblastic elements predominating and undergoing malignant degeneration, transforming the tumour into an adenocarcinoma. Epiblastic elements are scanty, but help to establish the teratoid nature, and link this specimen up with the next. Specimens of this type illustrate the important fact that teratoid tumours exhibit various gradations between the typical tumour containing all three blastodermic elements and those formed apparently entirely of one element.

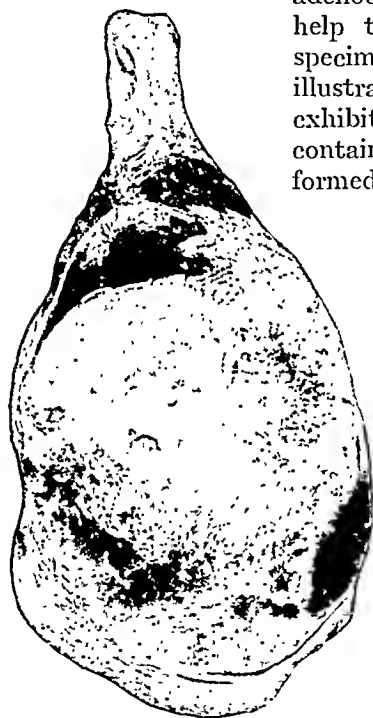


FIG. 19.—*Teratoma testis*: Hypoblastic (adenocarcinomatous) overgrowth. The cut surface is of 'medullary' type, with hemorrhages. The testis is not visible to the naked eye but persists microscopically as a peripheral band. The epididymis is fused.

**MACROSCOPIC.**—One half is preserved, the complete tumour being the size of a small melon. The cut surface presents a brain-like appearance with several circumscribed paler nodules, and there are numerous small hemorrhages throughout the tumour substance. There is a small hæmatocele at the upper pole, and the lower part of the tunica vaginalis is obliterated by the adhesion of the tumour to the scrotum, which probably accounts for the inguinal metastases. The epididymis is fused and indistinguishable (Fig. 19).

**MICROSCOPIC.**—*Hypoblastic Elements.*—The tumour is a vascular adenocarcinoma derived from columnar cells and supported by a scanty fibrous stroma (Fig. 20).



FIG. 20.—*Teratoma*: Cylindrical or columnar adenocarcinoma, showing spaces from which the tumour has arisen.

*Specimen 8.*—*Hypoblastic overgrowth*: (c) Pure adenocarcinoma.

This specimen was removed from a man, age 42, who gave a six months' history of swelling of the testicle with recent enlargement of the inguinal glands.

The columnar or cylindrical-celled character is displayed to great advantage in the cells lining the spaces and in the cross-sections of the papillae, which show the cylindrical cells arranged around a fibrovascular core. Considerable areas of new growth have undergone degeneration and hæmorrhagic infiltration and stain poorly. Glandular spaces lined by columnar cells retaining a simple character appear on the surface of parts of the tumour, show a transition into cancer, and may certainly be regarded as the starting points of the cancer.

*Epiblastic and Mesoblastic Elements* do not appear in the sections examined.

*The Testis.*—The degenerated remains of the testis can be recognized as a peripheral band at the surface of the tumour.

*Comment.*—The specimen is to be regarded as a glandular carcinoma derived from the hypoblastic elements of a teratoma with suppression of the other blastodermic derivatives, though of course it is possible that further sections might reveal traces of the other layers. The essential fact, however, is that the tumour is predominantly an adenocarcinoma of a structure sometimes exactly duplicated in indubitable teratoid tumours by malignant conversion of the hypoblastic elements.



FIG. 21.—*Teratoma*: (Left) Spheroidal-celled cancer of scirrhus type. (Right) Columnar-celled glandular elements giving origin to the cancer.

*Specimen 9.*—*Hypoblastic overgrowth*: (d) Spheroidal-celled scirrhus carcinoma.

Spheroidal-celled carcinoma is sometimes regarded as a special type, and consequently a short description of the following typical example appears warranted. By some it is related to a specially early age incidence—26·3 years (Schultz and Eisendrath)—and the present case happens to conform to this.

T. W., age 27. Swelling of the left testicle noticed for four months. It steadily grew, and on admission to hospital the tumour had reached the size of the foetal head, fungated through the scrotum, and involved the left inguinal glands. In spite of an extensive removal of tumour, scrotum, and glands, the patient returned in five months with large metastatic tumours in the upper abdomen.

**MICROSCOPIC.**—The cut surface is of the 'medullary' type, and the epididymis is incorporated and indistinguishable.

**MICROSCOPIC.**—The bulk of the tissue examined presents the typical appearances of spheroidal-celled cancer, with here and there indications of the glandular elements giving rise to the tumour (Fig. 21). Parts of the neoplasm are scirrhus,



and scanty epithelial cell-groups are separated by a considerable amount of fibrous tissue, but other areas are encephaloid. Earlier sections showed the spheroidal type of cell almost entirely, and the invasion of the inguinal glands has the same character, but later sections demonstrated abundant areas of columnar-celled adenocarcinoma and simple glandular spaces, and indicated the origin of the spheroidal elements. In addition there are some large spaces lined by cubical or columnar cells, which contain deeply staining colloid material and resemble thyroid tissue.

*Comment.*—The tumour is a carcinoma, spheroidal-celled elements predominating, and arising from an overgrowth of hypoblastic derivatives. Its teratoid origin is indicated by the mixed glandular characters and by the existence of a peripheral band of degenerated testicular substance. The brief duration and comparatively large size of this tumour, in common with the two preceding specimens, should be noted as indicating the high degree of malignancy and rapid course of the teratoid glandular cancer.

#### TERATOID CARCINOMA OF GLANDULAR ORIGIN, PROBABLY HYPOBLASTIC.

This group has already been partly considered in a previous section and its wide scope indicated. All that is necessary here is to elaborate the carcinomatous conversion of the hypoblastic elements of teratoid tumours and to indicate the relations of this group to the specific germinal-celled tumours to be described later. Examination of a large number of teratoid tumours demonstrates the numerous variations in type of the hypoblastic elements, and often reveals early cancerous change in apparently innocent tumours (*Figs. 22, 23, 24, and 25*). In many cases the hypoblastic origin is readily traced, but in others it is possible that the malignant transformation has begun in glandular structures of epiblastic derivation—mammary tissue, for example. Consequently it is safer to speak of teratoid carcinoma of glandular epithelial origin, though in the majority of cases it is the hypoblastic structures which are at fault.

**Types of Carcinoma.**—It is traditional to describe carcinomatous tumours of the testis according to the character of the cells and their arrangement. Thus, cancers are described as columnar-celled, spheroidal-celled, alveolar, scirrhus, and encephaloid. Little importance should be attached to these sub-divisions, for though a testicular tumour may be predominantly spheroidal-celled, cubical or columnar elements are usually demonstrable, and as in the breast, for example, parts of a tumour may be scirrhus and others encephaloid. This method of grouping, though artificial and having no special histological significance, has the merit of convenience. The following types of teratoid carcinoma of glandular and probably hypoblastic origin may be distinguished, but are frequently merged: (1) *Columnar- or cylindrical-celled cancer*; (2) *Spheroidal-celled cancer*; (3) *Undifferentiated more or less round-celled cancer*.

**COLUMNAR-CELLED CANCER.**—This is a frequent type, and the examples appearing in the present series reproduce all the features of adenocarcinoma as observed in other parts of the body. Though it may appear in pure form as the result of one-sided overgrowth, the facts that similar glandular elements are seen characteristically in teratoid tumours, and that some specimens contain mixed elements, seem incontrovertible evidence of its essential teratoid nature.

**SPHEROIDAL-CELLED CANCER.**—This is also a fairly common type, and for the same reasons is generally regarded as arising from the epithelial elements of a teratoma, and its accredited early age incidence in the twenties supports



FIG. 22.—*Teratoma*: Carcinomatous transformation of glandular elements, with two nodules of stratified epithelium.



FIG. 23.—*Teratoma*: Carcinomatous transformation of glandular elements. Other portions of section show simple spaces lined by cubical cells, also stratified epithelium and cell nests.



FIG. 24.—*Teratoma*: Carcinomatous transformation of glandular elements. Near centre, spaces lined by columnar cells which in other parts are infiltrating the stroma.



FIG. 25.—*Teratoma*: Carcinomatous transformation of glandular elements. An undifferentiated round-celled type of carcinoma, resembling sarcoma and spermatocytoma, but definitely originating from spaces lined by cubical cells as seen in other parts of the tumour.

this view. It reproduces exactly the features of spheroidal-celled cancer as observed in the breast and other organs.

**UNDIFFERENTIATED ROUND-CELLED CANCER.**—It is not uncommon to observe in teratoid tumours areas of round-celled cancer closely resembling sarcoma and yet arising undoubtedly from glandular tubes or spaces (*Fig. 25*). This presumably is a proliferation of cells of an embryonal type which lose all differentiation. The recognition of this variety is obviously important, as it may resemble the specific tumours of germinal-celled origin and is likely to be confounded with them.

As far as can be judged from the present group of teratoid tumours, this type of carcinoma has appeared only in a patchy form, and an origin from glandular spaces of an embryonal character has been readily traced, if not in the same section, in others taken from the same tumour. Yet, if we allow full scope to the principle that a one-sided overgrowth often occurs in teratoid tumours and that such overgrowth is more frequent in the embryonal elements, it would seem highly probable that pure or almost pure embryonal carcinomata of teratoid origin may occur from time to time. Such a cancer would closely resemble the germinal-celled tumours to be described in a second paper, and in the absence of mixed elements might be extremely difficult to differentiate from them.

#### EPIBLASTIC OVERGROWTH.

**The Teratomatous Cyst (Dermoid Cyst) of the Testis.**—The testicular 'dermoid', as might be expected, presents general resemblances to the better-known ovarian dermoid cyst, with the remarkable difference that it is so rare that only some half-dozen cases have been recorded in Great Britain within recent times. The pioneer work of Verneuil<sup>4</sup> probably accounts for the more frequent reference to it in French literature. The writer has met with only one example from the human subject in the Edinburgh collections. On the other hand, the dermoid is a comparatively common tumour in the ovary, while the polycystic teratoma is rare if the common multilocular cystadenoma is excluded from the teratoid category.

No satisfactory explanation of this curious reversal of the incidence of the two types in the testis and ovary is forthcoming, though it is well known that the epiblastic elements are relatively better represented in the ovarian teratomata. Nicholson<sup>5</sup> relates it to the difference in pressure exerted upon the developing tumour in the seminiferous tubule and in the Graafian follicle. In the former, pressure limits development and causes irregular growth, and the resultant tumour is of the small polycystic variety, while in the Graafian follicle the comparative absence of pressure allows greater development, and the single cyst predominates and may attain considerable dimensions. Both testis and ovary, however, produce intermediate types or links between the typical dermoid and the polycystic teratoma, and Barrington<sup>6</sup> reports a tumour of the testis in which a dermoid cyst representing the epiblastic elements was associated with a solid carcinoma evidently derived from columnar hypoblastic elements.

The term 'dermoid cyst' of the testis should be discarded, as it indicates only the striking and obvious feature, and gives no indication that the cyst is really a localized epiblastic overgrowth of a teratoma, as the co-existence of mixed elements and the occurrence of intermediate types conclusively

proves. Furthermore, it may lead to confusion with the skin dermoids that occasionally develop in the median raphé of the scrotum and have no relation to the testis proper.

Two examples are here described. The first, which is preserved in the Museum of the Edinburgh Royal College of Surgeons, I owe to the courtesy of C. W. Maegillivray, who removed it from a patient, age 25, in the Royal Infirmary, Edinburgh. The second, a cyst of the testis of a horse, is preserved in the University Anatomical Museum, and is briefly described for a special reason.

Teratomatous cysts of the human testis are so rare that no apology is needed for describing the present highly typical example. The *clinical history* is graphically given in Mr. Maegillivray's own words, and illustrates certain characteristic points discussed below :—

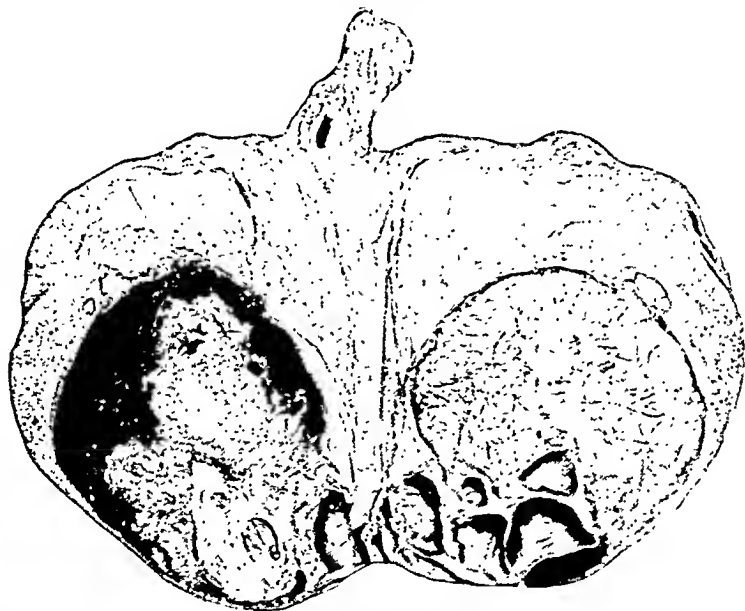


FIG. 26.—*Teratoma testis*: Epiblastic overgrowth. Teratomatous (dermoid) cyst surrounded by testicular substance and containing sebaceous material encmeshing hair. The foetal rudiment is best seen in the left half.

*Specimen 10.*—"A. W., 25, single, fisherman. Left testicle unduly large since birth. Indeed, the mother of the patient used to encourage neighbours to come and see it as a curious sight. It 'grew as he grew', and gave him no trouble except for its being large. At the age of three he was taken to the Children's Hospital about it, and his mother was told to bring him back next day, which she did not do. Has had gonorrhœa and sores on the penis. Came to the Royal Infirmary, Edinburgh, in June, 1900, to seek advice because he contemplated matrimony. The left testicle was as large as a man's fist. Dermoid cyst excised, June 13. Discharged cured June 27, 1900."

**MACROSCOPIC.**—The testicle and enclosed cyst form an ovoid tumour the size of the foetal head. The testicular substance appears to be increased in amount, and is disposed as a thick peripheral layer completely surrounding the cyst, and the epididymis is stretched over it. The tuniæ albuginea and vaginalis are closely

apposed and in part adherent. The wall of the cyst is slightly pigmented and obviously of the nature of skin, and is covered with hair embedded in a mass of fatty or sebaceous material. The fœtal protuberance (Rokitansky's rudiment) appears as a large projection in its lower part, and no teeth are present (*Fig. 26*).

**MICROSCOPIC.**—The cyst wall examined is of the nature of fully-developed skin, with hair, sebaceous, and sweat follicles (*Fig. 27*). Resort has been had to the X rays to demonstrate the existence of mixed elements in the form of bone in relation to the fœtal rudiment (*Fig. 28*).



FIG. 27.—*Teratoma*: Epiblastic overgrowth. Wall of teratomatous cyst near fœtal rudiment, showing skin structure.



FIG. 28.—Skiagram of left half of same specimen, showing irregular pieces of bone beneath the fœtal rudiment. The right half contained no bone and is not reproduced.

**Comment.**—This tumour illustrates the following points relative to the teratomatous testicular cyst, and these are shortly compared with those exhibited by the corresponding tumour of the ovary.

1. The tumour is congenital and is present at birth, a feature exhibited by nearly all the recorded cases, as Bland-Sutton<sup>7</sup> points out. The tissues of the teratoma in this case are coeval with those of the host and attain full development, while the less developed teratoid tumours probably arise later in life and their tissues remain more or less embryonal. The ovarian cyst, owing to its concealed position, usually escapes notice till after puberty, and the patient commonly appears for operation between the ages of twenty and thirty.

2. It conforms in every essential detail with the gross structural features of the ovarian tumour, the cyst appearing in the midst of the testis and expanding it in the form of a peripheral capsule as growth advances and the skin secretions accumulate and the hair grows.

3. The tumour 'grew as he grew', causing inconvenience only by its bulk, and showing no signs of malignancy clinically or histologically. The more adult the structure of a teratoma the less prone is it to malignant change,

but malignant conversion of the skin elements has been recorded rarely in connection with the ovarian teratomatous cyst and indicates the narrow line between the simple and the malignant in teratoid tumours of the sex glands.

4. The foetal rudiment displays no teeth, so often seen in the ovarian tumour, but this accords with the relatively poorer epiblastic representation in teratoma testis generally.

5. The foetal rudiment has related to it bony structures, indicating the essential multiple-layered nature of the tumour. The ovarian cyst is well known to have aggregated beneath the foetal rudiment a collection of mixed tissues such as cartilage, bone—often bearing teeth—muscle, and nervous elements; and by analogy we may reasonably assume their existence in the testicular cyst. In any case the presence of bone is indubitable, establishing the essential teratoid nature of the so-called dermoid, which the presence of the striking skin-lined, hair-clad cyst is apt to dwarf.

*Specimen 11.*—Multilocular teratomatous cyst of horse's testis.

No history is attached.

**MACROSCOPIC.**—The testis and enclosed cysts form a tumour the size of a large orange which is split sagittally and folded apart. This makes it difficult to say exactly how many skin-lined cysts are present, but there is certainly a large central one, and two (probably three) smaller ones lying more peripherally. The large cyst is the size of a small egg, and shows at one part a small projection or foetal rudiment overlying a mass of hard bone, and all display tufts of coarse hair resembling mane or tail. Again, no teeth are present. A small amount of sebaceous material is embedded in the roots of the hair (*Fig. 29*).

The testis is massed chiefly at the upper pole, but exists all round as a circular layer enclosing the cysts. The epididymis is spread over the back of the tumour.

**MICROSCOPIC.**—The lining of the cysts is of the nature of well-formed skin displaying the usual appendages. The existence of mixed elements in the form of masses of bone is evident at a glance, and is demonstrated by a skiagram (*Fig. 30*). The testicular substance is normal as regards the number and shape of the seminiferous tubules, but the cells are degenerated, probably owing to the formalin fixation.



**FIG. 29.**—*Teratoma testis (horse)*: Epiblastic overgrowth. Multilocular dermoid cyst, showing skin-lined cysts and tufts of hair resembling mane or tail. A crescent of bone is seen in the centre, and the testicular substance appears chiefly at the upper pole.

**Comment.**—The specimen is obviously a teratoma, as the presence of mixed elements indicates, and is an example of overgrowth of the epiblastic elements reaching a high stage of development. Bland-Sutton states that teratomatous cysts of the testis are not uncommon in the horse. The teratomatous cyst is typically single, and this specimen has been described

because the skin-lined cysts are multiple. This type may be regarded as a link in the chain between the highly developed single-cysted variety (teratoma) and the less developed multiple-layered tumour (teratoid).

Multilocular dermoid cysts of the ovary have been occasionally recorded, and presumably indicate a development from more than one germinal cell,

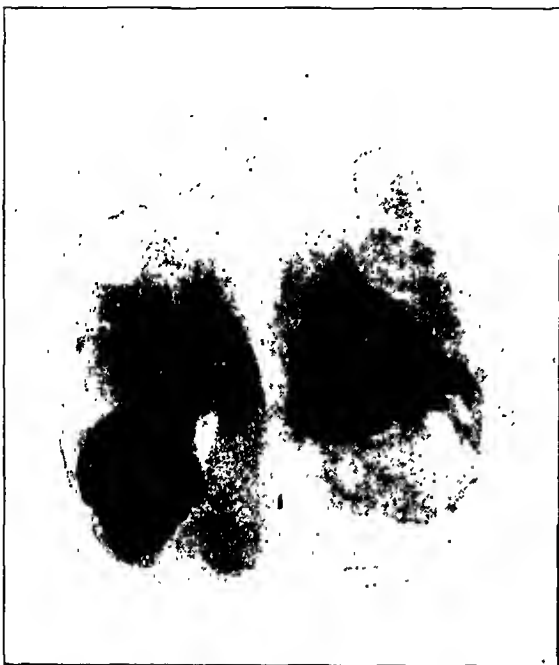


FIG. 30.—Skiagram of specimen *Fig. 29*, showing masses of dense bone in relation to the skin-lined cysts.

resulting in the production of several abortive embryos. This would appear to be true of the fully-developed cyst with other structures in close relation to it, but any epidermoid cyst as seen so often in the simpler teratoid tumours might attain full development, without any limit to multiplicity, yet arising from a single germinal cell.

*Specimen 12.*—Epiblastic overgrowth: Basal-celled carcinoma.

No history is attached to this specimen, which belongs to the Spence collection and is labelled a fibrosarcoma.

**MACROSCOPIC.**—The tumour is bisected and is the size of a grape-fruit. The cut surface presents a solid or even fibrous appearance and is divided into lobules by broad fibrous septa in which the cut ends of numerous vessels are visible—in short, a surface inspection suggests that

the condition is fibrous or fibrosareomatous. The epididymis is not involved.

**MICROSCOPIC.**—The first section showed almost entirely the features of a fibroma or fibrosareoma and appeared to bear out the existing diagnosis. There were, however, scanty groups of darkly stained small spindle or columnar cells displaying an alveolar arrangement and suggesting epithelial structures. Later sections revealed the significance of these, and demonstrated the presence of a basal-celled carcinoma resembling rodent cancer (*Fig. 31*). Sections taken from superficial areas show a covering peripheral band of testicular tissue separated by a capsule from the underlying characteristic irregular masses of basal-celled cancer, which are supported by a large amount of fibrous or even fibrosareomatous tissue. At the periphery of the cancerous groups the cells are arranged in the form of spindles of a definitely columnar appearance, giving a superficial resemblance to columnar-celled cancer, while the central cells are mostly oval. A characteristic feature displayed by the cancerous masses is the presence of numerous vacuoles bound by columnar or spindle cells. This is the result of mucous or myxomatous degeneration, and produces a spurious adenomatous appearance, to which the term 'reticulated epithelioma' is sometimes applied. Further from the surface the cancer cells are less numerous, and the supporting embryonal fibrous (sarcomatous ?) matrix becomes so abundant that in some fields it is the sole tissue represented. It is interesting to observe in places that the spindle or oval epiblastic cells merge into, and appear to take part in, the formation of the stroma.

As in basal-celled cancer of the skin, horny changes and cell-nest formations are absent.

*Comment.*—The tumour is a basal-celled carcinoma reproducing the features of typical rodent cancer, and the abundance of the embryonal fibrous tissue suggests that we may be dealing with a combined tumour of epiblastic and mesoblastic origin. No clue is given to the origin of the carcinoma, but it probably arises from the epiblastic elements of a teratoma, the fibrosarcomatous (?) tissue representing mesoblastic elements. The existence of a peripheral band of testicular tissue goes to confirm the teratoid nature.

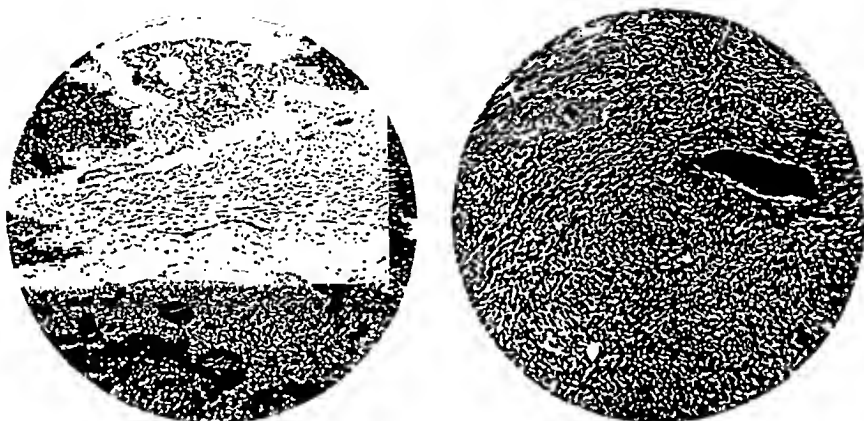


FIG. 31.—*Teratoma*: Epiblastic overgrowth. Basal-celled carcinoma. (Left) Typical rodent ulcer. (Right) Fibrosarcoma.

#### OTHER TYPES OF MALIGNANT EPIBLASTIC OVERGROWTH.

It is generally accepted that teratoma testis differs from the corresponding tumour of the ovary in the relatively poor representation of the epiblast, and consequently it is difficult to bring forward examples of malignant epiblastic overgrowth, though it is possible that some of the glandular carcinomata occurring in this series which have been classed as hypoblastic in origin are really derived from epiblastic epithelial elements.

Malignant epiblastic tumours of the testis may be assumed to occur in the following forms: (1) *Squamous epithelioma*; (2) *Basal-celled carcinoma*, already described; (3) *Neuro-epithelial tumours*; (4) *Chorion-epithelioma*.

Types (1), (3), and (4) do not occur in the present series, but a consideration of the literature supplies examples, and to make this survey more complete a brief consideration of these types appears warranted.

**Squamous Epithelioma.**—No example of this tumour in anything like a pure condition has appeared in the present specimens, which makes the discovery of the basal-celled carcinoma all the more remarkable. At the same time it is not uncommon to find areas in teratoid tumours where the epiblastic structures show a local preponderance, and in one specimen squamous epithelioma of unknown origin was suspected by a previous investigator.



In two old specimens epiblastic elements figured largely, but this was probably due to the greater resistance to disintegration provided by the horny tissues (*Fig. 32*).

The writer finds it difficult to appraise the value from the point of view of malignancy of the commonly observed epidermoid cysts lined by squamous epithelium and the equally common cell-nest or epithelial pearl formation. Though cell-nest formation is a highly typical feature of ordinary squamous epithelioma, it would appear that the epidermoid cysts so commonly present in teratoid tumours provide a condition *par excellence* for the development of cell-nests quite apart from malignancy.

**Neuro-epithelial Growths.**—No example of genuine overgrowth of neuro-epithelium has been observed, but at the same time no particular search has been made for it. Small groups of ganglion cells and scanty neuro-epithelial tissue have occurred in two specimens. Ewing states, however, that neuro-epithelial cell groups occur frequently and may show malignant degeneration.

**Chorion-epithelioma.**—This highly interesting variation of teratoma testis, which reproduces in some cases, but not in all, the characteristic feature of chorion-epithelioma as observed in the female generative organs, is now a well-recognized form of testicular new growth, and is presumed to arise, if not from foetal chorionic trophoblastic structures, at least from epiblastic cells homologous with those of the chorion.

In genuine cases chorion-epithelioma is characterized macroscopically, as in the uterus, by the predominance of blood-clot, which may appear to compose the bulk of the neoplasm or may appear as a localized hæmorrhagic



FIG. 32.—*Teratoma*: Epiblastic overgrowth, showing concentric horny nests which form the greater part of this section.

nodule; and microscopically, by the multinucleated syncytial masses and the clear Langhans' cells. It also exhibits the same predilection to spread by the blood-stream and to form metastases in the lungs and liver. Nicholson<sup>8</sup> gives details of four chorion-epitheliomata met with in his collection of some sixty neoplasms of the testis, all occurring as a special development in teratoid tumours, and it is noteworthy that none appears in the present relatively large series, though it is highly possible that a more complete examination might have revealed examples. In one adenocarcinoma, multinucleated plasmodial masses were observed in several small foci scattered amongst the carcinomatous tissue, but were not typical of chorion-epithelioma.

This tumour has passed through several phases of interpretation, and it would appear that pseudo-chorion-epitheliomatous structures occur occasionally and complicate the question. Several cases of this type have been recorded. MacCallum,<sup>9</sup> for instance, figures a typical example associated with a

teratomatous tumour of the testis which invaded the inferior vena cava through the spermatic and renal veins. Hydatidiform, grape-like structures hung free in the circulating blood, and more or less filled the great venous channels, but in spite of the resemblance to hydatidiform mole the villus-like bodies were not covered by chorionic epithelium.

Certain French observers consider the syneytial masses as epithelial giant-celled formations developing around vascular spaces, and regard the condition as angiosarcomatous. These pseudo-types, however, do not invalidate the fact that genuine chorion-epitheliomatous tumours occur in the testicle, as Schlagenhauser<sup>10</sup> maintained, and reproduce all the characteristics of the uterine tumour—both local and metastatic—even occasionally manifesting the features of pseudo-gestation in the male breast.

### METASTASIS IN TERATOMA TESTIS.

Metastasis may take place by lymphatic spread, blood spread, or both, depending on the predominance or admixture of carcinomatous or sarcomatous elements. In addition it is possible that transplantation of tumour fragments may take place in comparatively innocent tumours, such as the chondromatous teratoma, by direct invasion of venous channels. Paget's enchondroma is often regarded as an example of this, and the secondary tumours in the lung as produced by transplantation after erosion of the spermatic vein.

**1. Lymphatic Spread.**—In the majority of cases carcinomatous tendencies prevail and metastases involve the lymph nodes, especially the lumbar group primarily, and the intrathoracic nodes, brain, kidney, etc., secondarily. Bland-Sutton<sup>11</sup> has described a cystic teratoma which was followed later by a similar tumour in the left supraclavicular glands—an evidence of spread by the thoracic duct. In several of the present series the corresponding inguinal glands were involved, but this has always been associated with fungation of the tumour through the scrotum, or with firm adhesion of the tumour and its membranes to the scrotum.

**2. Blood Spread.**—In some cases sarcomatous features prevail and produce metastases, especially in the lungs and liver. Cases are recorded in which a multiple-layered teratoma has been associated with pulmonary or hepatic metastases exhibiting purely a round-celled sarcomatous structure, and the same feature has been observed in the malignant teratomata of the ovary. Chorion-epithelioma in particular, like the corresponding tumour in the uterus, spreads by the blood-stream and produces pulmonary and hepatic metastases.

As Ewing<sup>12</sup> points out, the structure of the metastases raises several interesting points bearing on the growth potentialities of the cell components of the teratomata. Representatives of all three blastodermic layers may be included in the same metastasis, in which case it seems conclusive that the metastasis must have arisen from a cell or cells capable of producing all three layers, while in other cases individual metastases from the same tumour may reproduce individual layers of the teratoma, suggesting that each metastatic embolus has been endowed only with the limited capacity of reproducing its own kind, either in embryonic or adult form.

## AGE INCIDENCE.

Teratoma testis may be present at birth, as in the so-called dermoid, and thereafter may come to light at almost any age. Apart from the teratomatous cyst, there is in the present group a carcinoma of teratomatous origin from a boy, age 5. Most cases have appeared in the twenties, both of the comparatively innocent teratoma and malignant variations of it, and another group seems to be related to the forties in particular. It is impossible, however, to give any detailed information as to the age incidence, owing to lack of the requisite data in the majority of the older specimens. Only certain general impressions may be culled from the data available, and these suggest that a teratoma, innocent or malignant, may be associated with the full blast of adolescence (like the odontomata), or may lie latent till middle life (like the branchiogenetic cancer or cancer generally), when a predominantly carcinomatous tendency manifests itself.

## THE ORIGIN OF TERATOMA TESTIS.

This may still be considered speculative. For the purposes of this paper only a brief survey of certain theories is indicated, and the reader is referred to the well-known text-books of pathology for detailed information and discussion.

Teratoma testis may be assumed to arise:—

1. **From Spontaneous Development** of the ordinary sex or germinal cells of the testis by a process analogous to parthenogenesis. This conception is specially connected with the name of Wilms, and is backed to some extent by experimental evidence, for in the case of frog's ova it has been shown that an artificial stimulus may initiate segmentation and produce imperfect embryos. If we endeavour to postulate the artificial stimulus in man, injury would appear to be the most likely factor, for the testes—especially in youth—are liable to traumata, and the imperfectly descended testis which is credited with a special tendency to become malignant is not exempt from repeated strain or slight injury.

2. **From an Isolated Blastomere:** (a) **Primordial**; (b) **Germinal**.—

a. It has been shown experimentally that in the early stages of segmentation of the fertilized ovum, if one of the blastomeres is isolated and placed in a suitable medium, it may develop into a more or less well-formed embryo, for the primordial blastomere, like the fertilized ovum, is totipotent and capable of giving rise to cells of any order, and therefore to teratoid tumours.

b. The germinal blastomere or primitive germinal cell, the progenitor of the ova and spermatozoa, arises later in the segmentation of the ovum, and is also totipotent. We do not know the precise origin of the primitive germinal or sex cells. They are believed by some to develop from the specialized epithelium (mesothelium) covering the genital ridge, and by others to be special cells budded off the morula which migrate into the genital area. This latter view affords a satisfactory and common origin both for the genital teratomata and the rare extra-genital tumours situated in the thorax or abdomen,

for it is easy to suppose that a primitive sex cell (germinal blastomere) may miss its correct destination in the future sex gland and become isolated in the thorax or elsewhere, and develop into a teratoma. Even if it reaches its proper destination in the future ovary or testis, it may still fail in the case of the testis to assume an orderly arrangement within the seminiferous tubules. Then, instead of passing through the phases of the sexual cycle and ultimately producing spermatozoa, such a misplaced sex cell may segment and, in virtue of its multipotent characters, produce a teratoid tumour. If development proceeds *pari passu* with that of the host, an adult teratoma results, for the tissues of the tumour are coeval with those of the host (e.g., the teratomatous or dermoid cyst); while if the aberrant blastomere lies latent in the genital gland and only begins to develop at a later period, say at puberty or still later under the stimulus of an injury, a less complex teratoid tumour is likely to result, owing to a reduction in the growth potentialities of the aberrant sex cell with repeated division.

From a consideration of the above theories certain points appear to stand out. Of the 'parthenogenetic' conception little is known unless it be interpreted in terms of trauma. Of the aberrant primordial blastomere we again know little, but it is asking too much of it to expect it to find its way into the sex glands with such regularity as indicated by the remarkable frequency of teratoma in the ovary and testis as compared with other situations. Consequently, it seems highly probable that the point of origin of the teratoma must be a cell which is commonly present in the sex glands. In the case of the testis we are left with the isolated, aberrant germinal blastomere or primitive sex cell which becomes misplaced in the tissues of the testis, and the primitive sex cell which, following its ordained course, becomes included within the seminiferous tubules and later passes through the phases of the sexual cycle. There does not appear to be any vital difference between the orderly cell and its descendants, and the misplaced cell, except in the degree of potentiality, and such differences may perhaps be related to the various degrees of complexity exhibited by teratomata.

As regards the possibility of misplacement of sex cells in the ordinary course of development of the testis, it is most likely, as Ewing stresses, to occur in or near the rete where the seminiferous tubules should normally fuse with the excreting tubules. Such a likely site of misplacement accords with the frequent origin of teratoma testis in the mediastinum.

### CONCLUSIONS.

1. The majority of testicular tumours belong to the teratoid category.
2. The relative representation of the various blastodermic derivatives varies greatly in different specimens.
3. Overgrowth of one blastodermic element with partial or total (?) suppression of the others occurs frequently. Hypoblastic overgrowth is most common, mesoblastic next in frequency, and epiblastic least common.
4. Such overgrowth may be simple or malignant, and produces many variations from the typical three-layered teratoma.
5. Teratoma testis is potentially malignant to a high degree.

6. Many carcinomata of the testis can be directly related to the malignant conversion and overgrowth of the epithelial, more especially the hypoblastic, elements of teratomata.

7. The malignant change may be selective, but may affect both epithelial and connective-tissue elements in the same tumour.

8. The more adult the structure of the teratoma, the less liable is it to malignant change, and conversely.

#### FURTHER CONSIDERATIONS.

1. The fact that the bulk of the testis proper is usually situated towards the upper pole in teratoid tumours suggests that, in early cases at any rate, some diagnostic use might be made of it. Testicular sensation limited to the upper pole might occasionally give a clue to the nature of a particular tumour.

2. In cases coming under observation early, it would appear possible, from a study of the cut surface of the smaller tumours, to enucleate the teratoma from its capsule, leaving the testis and epididymis intact. However, in a paired organ like the testis such conservatism would rarely be indicated.

3. The use of the X rays may occasionally give additional information in a dubious serotal tumour if the cartilaginous elements are well developed, and especially if bone is a prominent feature, as in the teratomatous (dermoid) cyst.

The photographic work has been done by Mr. Frank Pettigrew, Technical Assistant to the Department of Surgery, to whom I am greatly indebted for his skill and care.

*(To be followed by an article on the Spermatocytoma Group.)*

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## THE AFTER-RESULTS OF OPERATIONS FOR MALIGNANT DISEASE OF THE BREAST.

By ALAN C. PERRY, London.

THE object of this investigation was to attempt to follow a relatively small series of cases through their subsequent histories in the hope that it might be possible to determine some or any of the factors which have a bearing on the prognosis after operation. It might appear presumptuous to draw conclusions after the recent monumental publication by the Ministry of Health<sup>1</sup> on this subject. The material, however, was collected some time ago, and it was felt that some details might be more evident on a close study of a small number of cases, although broad general conclusions could not be drawn. It is generally accepted, perhaps often without sufficient evidence, that prolongation of life results from the extensive modern operation performed for carcinoma of the breast. The excuse for this paper is that it gives a detailed analysis of the after-results in 653 cases, and attempts to correlate these results with possible factors affecting the prognosis in such cases.

**Prognosis without Operation.**—Before one can determine the value of any particular operation it is necessary to know exactly what would happen to the patient in the absence of any treatment. Therein lies the difficulty, as the ability to state with certainty how long any case of carcinoma of the breast will survive without operation is beyond the bounds of possibility. Furthermore, to ascertain even the stage reached by the disease on presentation for treatment is no easy task.

First, it is impossible to state how long the carcinoma has been present, for the patient's statement is notoriously inaccurate, as witness the number of cases who discover a fairly large lump by accident. Schwarzkopf<sup>2</sup> and Primrose<sup>3</sup> state that fourteen months is the average time that elapses between the known onset of the growth and the performance of any operation. The latter states that only 8 per cent of cases seek treatment within a month of the discovery of the lump, and that 50 per cent are untreated for a whole year. Deaver<sup>4</sup> gives the average history before operation as thirty months. This interval, so variously stated by these authorities, represents roughly the history of an operable case before coming for advice.

Secondly, the type of growth, in a clinical sense, has a great influence on the lethal rapidity. An 'acute' breast carcinoma will kill a patient within six months, whereas a slowly-growing one in an elderly patient may remain with her for ten or fifteen years and does not appear appreciably to shorten life. In such extreme cases the value of any operative interference would be especially difficult to determine, yet all such must be included in the statistics. The presence of undetectable visceral deposits in any particular case will shorten life considerably, and when one remembers that they may be present with even a small and comparatively slow growth, the difficulty of deciding on the

effect of the operation is intensified. Sampson Handley<sup>5</sup> states that if visceral deposits are present, life is unlikely to exceed twelve months.

When we come to consider these facts the immense difficulties facing any one who desires accurate knowledge are realized. The past and future of any particular case being lost in obscurity, one can only suggest the effect of operation on the latter with considerable trepidation.

**Standard of Cure.**—The only true cure in this disease is a hypothetical one—viz., a patient has survived her normal expectation of life after operation, and a most minute post-mortem examination shows no signs of carcinoma in any organ of the body, and death occurs from some entirely different cause. Such a proof of cure is impracticable, as even the most careful examination might fail to reveal a minute deposit of carcinoma in an inaccessible organ, such as the medulla of a bone.

Recurrence many years after operation does take place, and is becoming more recognized as an essential feature of the disease. In this series is found a case with a first recurrence in the spine after fifteen years. Such a case immediately negatives any attempt to fix a time limit for the standard of cure. Volkmann's three-year limit as a criterion was soon found to be too brief. Other writers, when giving statistics, attempt to give their results after a fixed period, although in view of these late recurrences this must necessarily be fallacious. Bloodgood,<sup>6</sup> giving the statistics of the Johns Hopkins Hospital, states the condition of the patients after five years. Peck and White,<sup>7</sup> whose figures are regarded as amongst the most satisfactory, state that 39 per cent of their cases were alive after five years. The usual method in these series is to state the number of patients alive and well, or with recurrences, after such a fixed period.

In this paper it has been deemed advisable to adopt the method of determining how long each patient has lived. This was done because it is felt that a patient who develops a recurrence after eight years, although she may not be 'alive and well', certainly does give some indication of the efficacy of the operation.

**Operative Mortality.**—Of 653 cases, 11 died in hospital, thus giving a mortality of 1.7 per cent; of these, one died from infection, one under the anæsthetic, one from heart failure, and the remainder from pulmonary complications and pulmonary embolism.

### FACTORS WHICH MIGHT INFLUENCE PROGNOSIS.

Certain factors are generally accepted as having some bearing on the after-results of carcinoma of the breast.

**The Length of History.**—This has been touched on previously, and in view of its inaccuracy must be disregarded as throwing any light on the survival period after operation. A lump may have been discovered some years before or only a few hours, and in each case we are completely ignorant as to the duration of the carcinoma. The figures in this series are so misleading that it was felt no great advantage would accrue by recording them.

**The Age of the Patient.**—It is commonly stated that the younger the patient the worse the prognosis, and this bearing of age on the disease will be

analysed later. It must be borne in mind, however, that there is always a certain degree of hesitancy in diagnosing carcinoma in a patient under 40; and therefore treatment may not be furnished as expeditiously as it might in older patients. Again, younger patients are more liable to the general disturbances following pregnancy, and exacerbations of the growth which undoubtedly follow child-bearing. Furthermore, cases are rarer in the younger, and therefore figures based on small numbers are not of the same value. The average age of the cases (at the time of operation) in this series is 51·3 years, and they are distributed in the following manner:—

AGE	NO. OF CASES	AGE	NO. OF CASES
Over 80	.. 1	45 to 49	.. 122
75 to 79	.. 8	40 „ 44	.. 105
70 „ 74	.. 25	35 „ 39	.. 40
65 „ 69	.. 45	30 „ 34	.. 26
60 „ 64	.. 74	25 „ 29	.. 7
55 „ 59	.. 73	Under 25	.. 2
50 „ 54	.. 118		

There is thus a total of 646 in which the age is stated. It will be seen that the maximum incidence of the disease occurs between the ages of 40 and 54, and this is in accordance with most of the other organs affected with carcinoma.

**The Pathological Type of Growth.**—Many classifications have been devised for the various kinds of malignant disease occurring in the breast, and it is generally conceded that they vary in respect of their malignancy. Certain varieties, however, occur infrequently, and it is therefore advisable to be cautious in drawing conclusions, because in a small number of cases it may be that the other factors are the more important. In only two varieties of the classification adopted could no deaths be traced, viz., the papillary cystic and the intracystic carcinomata. It is well, however, to bear in mind that none of the cases in these groups has been traced over a lengthy period, the longest being six years.

Carcinomatous hyperplasia, although often classified as a pre-cancerous rather than as a malignant condition, is included, because two cases died of secondaries, one three and the other seven years after the operation, and in neither were the glands at first involved. One was 42 and the other 50. The effect of the pathological type of growth on the after-results is fully discussed in a later section.

**The Presence of Affected Glands.**—Deaver and McFarland,<sup>8</sup> in their exhaustive book, *The Breast*, state that the absence of involvement of the glands gives a 4 to 1 better chance in the post-operative prognosis. Microscopic examination of the glands removed in this series was carried out in a vast majority of cases. It was found that only 28 cases showing microscopic deposits survived for seven years or over, whereas 84 cases showing no deposits in the glands survived for the same period. This gives a 3 to 1 chance in favour of the cases where microscopy shows the glands free.

It is interesting to note that two cases showed tuberculosis of the axillary glands removed at operation. One, age 51, died after two years, and the pathological report on her growth was carcinoma simplex. The other, age



61, showed no recurrence after ten years, and microscopy showed a scirrhus carcinoma.

The presence of clinically enlarged glands is of little value in prognostication, as there were 36 cases of non-specific chronic inflammation in the axillary glands on microscopic examination. Of these, 6 were untraced and 16 survived for more than seven years. It would, therefore, appear that if chronic inflammation is demonstrated in the axillary glands, the prognosis is likely to be improved in the proportion of 33 to 7.

**The Effects of Preliminary Excision.**—In this series there were 30 cases which underwent preliminary excision, as out-patients, by the general practitioner, or for diagnostic purposes. Of these, only 6 survived the seven-year period, and it is interesting to give their particulars.

*Table I.*—CASES OF EXCISION SOME TIME BEFORE OPERATION.

AGE	PATHOLOGY	PERIOD ELAPSING BEFORE COMPLETE OPERATION	RESULT
60	Intraductular carcinoma	1 month	13 years, alive and well
33	Carcinoma simplex ..	3 weeks	9 years, died
61	Intraductular carcinoma	2 years	8 years, died
58	Scirrhus carcinoma ..	2 weeks	7 years, died
60	Papillary cystic carcinoma	4 days	9 years, died (other causes post mortem)
29	Carcinomatous hyperplasia	3 weeks	6 years, alive and well

It will thus be observed that of these cases 4 belong to pathological groups which are considered to be of the less malignant variety, and 3 of these are included in what ought to be termed 'cures'.

Of the 14 cases in which preliminary excision was performed immediately preceding the operation, 4 could not be traced, and 4 had not been done long enough to show seven years' freedom, although they are still without a recurrence. Of the remainder, 4 exceeded the seven years, of which one died after ten years from other causes. The results in the remaining 3 were as follows :—

*Table II.*—CASES OF EXCISION IMMEDIATELY BEFORE OPERATION.

AGE	PATHOLOGY	SURVIVED	RESULT
42	Scirrhus .. ..	4 years	Died : recurrence other breast
34	Scirrhus .. ..	1 year	Died : recurrence in spine
42	Carcinomatous hyperplasia	3 years	Died : carcinoma of lung

Few sound conclusions can be drawn from this small number of cases ; but it would appear that preliminary excision immediately followed by the complete operation has little effect on the question of prognosis, as either of the first two cases would have a grave prognosis. Regarding the third, there was a recurrence within thirty months in the supraclavicular glands,

and this would appear to suggest considerable malignancy in spite of the microscopic diagnosis.

Such evidence as we have would seem to indicate that preliminary excision at a period remotely antecedent to the complete operation, for diagnostic or other purposes, is a dangerous proceeding, whereas excision as a prelude to the operation has little or no influence on the after-result. This is, of course, in accordance with the accepted teaching.

**Selection of Cases.**—The results of any particular series will depend largely on the selection or otherwise of cases for operation. In this series, however, several surgeons have performed the operations, and each would have a different standard of operability. Furthermore, when we consider that cases who die from internal secondaries have a much more peaceful death than those who die with a fungating growth, it is unlikely that cases on the verge of being inoperable have been refused what at best might be only a palliative measure. It is certain, therefore, that many advanced cases are included. This is borne out by the fact that no fewer than 20 showed ulceration of the skin, and is further demonstrated when we find that 61 per cent of cases were adherent to the overlying skin.

Regarding private patients and hospital patients, there is said to be a more favourable prognosis for the former than the latter. The difficulty of following up hospital patients, particularly if they are well, must, however, be borne in mind. In the practice of one surgeon the private patients show 50 per cent of possible cases surviving the seven-years' period, whereas hospital patients show only 21 per cent.

**The Effect of X rays.**—An attempt was made to ascertain the effects of X rays applied to cases as an adjunct to operative interference. It was found extremely difficult to get comparable figures, because it is only in recent years that the extensive application of X rays has been practised after operation; and, furthermore, if X rays are applied after operation, there is no means of knowing that the operation was not entirely responsible for the absence of secondaries. Undoubtedly X-ray treatment of the secondaries developing in the scar was efficacious in numerous instances, with the almost certain prolongation of life. Therefore it must certainly be continued as a prophylactic as well as a means of treatment for recurrences.

**The Situation of the Growth in the Breast.**—The superficial lymphatics from the lower inner quadrant of the breast drain downwards to the epigastrium and so reach the abdominal lymph glands. This is the so-called dangerous area of the breast, because once a carcinoma in that region has invaded the skin, lymphatic spread to the abdomen is easy, and secondaries within the peritoneal cavity are probable and, of course, beyond treatment. Before one can determine this point, one must observe the relative frequency with which different parts of the breast are involved. In this series, where the situation of the growths is stated, we find the following percentages:—

Upper and outer quadrant	..	50 per cent
Upper and inner	..	17 "
Lower and outer	..	15 "
Lower and inner	..	6 "
Central	..	12 "

On analysing the cases that have survived for over seven years we find the situation represented in the following percentages :—

Upper and outer quadrant	..	46	per cent
Upper and inner	..	17	„
Lower and outer	..	12	„
Lower and inner	..	9	„
Central	..	16	„

There is thus very little difference between the two sets of figures, any slight advantage as to the prognosis being for the lower and inner quadrant or so-called dangerous area. It would be wrong to be dogmatic on this small series of cases; but it seems that the situation of the growth has very little bearing on the question of prognosis, and certainly the carcinoma in the lower and inner quadrant does not increase the danger to the patient.

### RESULTS OF CASES.

The cases examined for the purposes of this paper were operated on by certain surgeons up to within two years of the time that the investigation took place. Of the total number, 80 were completely untraced, and 182 were traced for a period and then lost sight of. The method adopted was to learn the length of time that the patient had survived the operation with or without recurrences. This was done for certain reasons instead of selecting all the cases operated on up to, say, five years ago, and ascertaining how many were alive and well. Firstly, a surgeon may have followed up a case for seven years, and it may be impossible to say what has happened after that time. Secondly, late recurrences do occur, and the fact of a patient being alive and well after five years is in no sense a cure, any more than the patient who develops a recurrence after ten years. Thirdly, it was thought that the early recurrences might throw some light on the type of case that gave rise to them, and also on the effect of treatment on such recurrences. Lastly, it might show when recurrences did occur, where they occurred, and perhaps why.

*Table III.*—RESULTS INCLUDING ALL UNTRACED CASES.

YEARS LIVED AFTER OPERATION	NUMBER OF CASES	POSSIBLE NUMBER OF CASES	PERCENTAGE
Over 16 ..	6	67	9
15 ..	9	69	13
14 ..	14	87	16
13 ..	22	141	16
12 ..	29	165	17
11 ..	39	195	20
10 ..	50	244	20
9 ..	65	297	22
8 ..	86	365	23
7 ..	112	395	28
6 ..	128	462	28
5 ..	153	553	28
4 ..	196	600	33
3 ..	236	638	37
2 ..	349	653	53

*Table III* includes all untraced cases, whereas the next table shows the partially traced and totally untraced cases separated out. The percentage and figures in *Table IV* are completely different from those in *Table III*, as the 'alive cases' are those alive in 1923, whereas *Table III* only takes cognizance of the number of years lived. For example, a woman may have lived for seven years and died five years ago; therefore she appears in *Table IV* as died, but in *Table III* she appears as having lived seven years.

*Table IV.*—RESULTS EXCLUDING PARTIALLY AND TOTALLY UNTRACED CASES.

Yr TO	TOTAL OPERATIONS	NOW DEAD		NOW ALIVE		PARTIALLY TRACED	UNTRACED
		Number	Per cent	Number	Per cent		
1907	67	18	27	6	9	27	16
1908	69	18	26	8	11	27	16
1909	87	25	29	10	11	36	16
1910	141	41	29	18	13	58	24
1911	165	55	33	23	14	60	27
1912	195	67	34	30	15	67	31
1913	244	89	36	45	18	77	33
1914	297	105	35	58	19	89	45
1915	365	130	36	77	21	103	55
1916	395	142	33	85	22	110	58
1917	462	164	35	106	23	128	68
1918	553	175	30	137	25	170	71
1919	600	187	31	174	29	171	72
1920	638	190	29	189	29	180	79
1921	653	198	29	193	29	182	80

In *Table III* it is interesting to note the sudden drop in the percentage of those surviving after seven years. The only deduction one can draw from this is that an eight-year survival period would be a useful one to adopt if it were desirable to establish any period as indicative of good results from the operation.

**The Effect of Age on Prognosis.**—*Table V* demonstrates that the most favourable age for operation is the quinquennium 50 to 54, 40 per cent of

*Table V.*—THE EFFECT OF AGE ON PROGNOSIS.

AGE	PER CENT LIVING MORE THAN 7 YEARS		AVERAGE NUMBER OF YEARS	PER CENT LIVING MORE THAN 3 YEARS		AVERAGE NUMBER OF YEARS
Over 70	..	—	9.0	44	3.9	
65 to 69	..	12	8.5	40	5.4	
60 „ 64	..	22	8.3	44	6.2	
55 „ 59	..	11	7.8	28	5.7	
50 „ 54	..	20	10.0	40	7.1	
45 „ 49	..	15	9.1	32	6.6	
40 „ 44	..	13	9.7	37	6.0	
35 „ 39	..	7	9.0	24	5.9	
30 „ 34	..	16	8.3	31	6.1	
Less than 30	..	—	—	15 cases	5.6	

such cases surviving more than three years, with an average of 7.1 years, and no less than 20 per cent of such cases surviving more than seven years, with an average of 10 years. It is interesting also to note that the results for ages 60 to 64 are better than those for ages 55 to 59. Can it be possible that immediately after the menopause a period ensues of lessening resistance to carcinoma, after which it rises again? These figures also bear out the statement that carcinoma has a much worse prognosis in the young, with the exception of the age period 30 to 34; but as the cases are rarer at this age, the figures are more unreliable than where larger numbers are concerned.

### The Influence of the Pathological Variety of Growth.—

1. *Scirrhus Carcinoma*.—This group formed 58 per cent of the whole series. Of these :—

37.5	per cent	lived more than 3 years,	with an average of 5.9 years
10.4	"	" " " 7 " " "	8.7 "
21	"	died within 3 years	" " "
14	"	were untraced.	

2. *Carcinoma Simplex*.—This group formed 14.2 per cent of the whole series, and of these :—

32.4	per cent	lived more than 3 years,	with an average of 7.8 years
16.2	"	" " " 7 " " "	11.2 "
19	"	died within 3 years	" " "
20	"	untraced.	

3. *Medullary Carcinoma*.—This group formed 8.8 per cent of the whole series, and of these :—

38	per cent	lived more than 3 years,	with an average of 7 years
21	"	" " " 7 " " "	9 "
19	"	died within 3 years	" " "
19	"	untraced.	

4. *Mucous Carcinoma*.—Only 4 cases were thus described, and one of these died after two years, one was untraced, and the others survived over three years.

5. *Papillary Cystic Carcinoma*.—Six cases were described, and no death was recorded.

6. *Intracystic Carcinoma*.—Twelve cases were described, with no deaths, although two cases were untraced.

7. *Intraductular Carcinoma*.—Eight cases, of which 2 were untraced; of the others, 2 died at nine and eight years respectively, and the others survived seven years or over.

8. *Carcinomatous Hyperplasia*.—Fifteen cases are included in the series, of which the large number of 5 were untraced; 2 died at seven and three years respectively, and the remainder are alive with an average survival of five years.

9. *Sarcoma*.—Only 3 cases were included, and of these one could not be traced, one is alive after seven years, and the other died after two.

The first and most striking feature of these figures is the large percentage of cases of medullary carcinomata that survive more than seven years, this percentage representing twelve cases, one of whom is alive and well thirteen

years after the operation. It would be rash to state that on these figures medullary carcinoma is less malignant than scirrhus carcinoma, but they certainly demonstrate the fact that medullary carcinoma has by no means a hopeless prognosis as is so often thought, but that the survival period may be considerable. Furthermore, this list demonstrates that carcinomatous hyperplasia in the breast is a definitely malignant condition and should be treated as such.

### RECURRENCE AFTER OPERATION.

There is definite evidence of recurrence after operation in 35 per cent of the cases examined. The situation of the first observed recurrence expressed as percentages was :—

Local (i.e., the operation area)	52 per cent
Supraclavicular glands ..	15 ..
Intrathoracic ..	14 ..
Intra-abdominal ..	10 ..
Spinal ..	8 ..

When we examine the time at which these recurrences first appeared after operation we observe :—

*In the First Six Months.*—During this period 14 per cent of the recurrences occurred, and the average age of the patients affected was 45·5 years.

*In the First Year.*—Before the twelve months had expired, 40 per cent of recurrences occurred, with an age incidence averaging 46 years.

*Within the First Eighteen Months.*—Here 55 per cent had occurred, with an average of 46·6 years.

*Within the First Two Years.*—70 per cent of recurrences had occurred, with an average age of 47·2 years.

*After Two Years* 30 per cent of the total number of recurrences first appeared, and these cases had an average age of 51 years.

No fewer than 9 cases developed recurrences after seven years, the longest period being that of the one previously mentioned at 15 years—spinal in situation.

Several cases which developed recurrences and were treated survived for considerable periods. One four years after the operation developed a secondary nodule which was excised, and she was in good health five years later. Another had a local recurrence three months after, was treated with X rays, and was alive and well five years later. A third developed a secondary after two years, and this was excised, but she died of general recurrences three years later. A fourth and very interesting case, age 33, developed after eighteen months a secondary which was removed, and she died six years later from abdominal recurrences.

From the investigation of cases developing recurrences after operation one arrives at certain deductions :—

1. Recurrence is a frequent post-operative event, occurring in 35 per cent of all cases, or rather more if untraced cases are excluded.

2. More than half of these recurrences are at first situated in the operation area. This would suggest that operators do not remove all the affected

tissues, which should be an argument for making the operation, even in early cases, as thorough as possible.

3. First recurrences in the supraclavicular glands occurred in 15 per cent. We might reasonably infer that excision of these glands should be practised in all cases, even though they form a remaining barrier between the growth and the thorax. No case of supraclavicular gland enlargement treated either by excision or X rays could be traced as having survived more than eighteen months, and intrathoracic deposits were frequently present before death.

4. Early recurrences are more common in younger patients, and late recurrences in the older. This would seem to confirm the conclusion previously reached, that carcinoma is a more malignant disease in patients before the menopause.

5. The treatment of local recurrences does not appear to be hopeless. Immediately they are detected they should be widely excised and further prophylactic doses of X rays administered. In such cases one might tentatively suggest that a local recurrence should be treated as the original disease was, and that the supraclavicular glands be excised, if this has not been already done.

6. X-ray treatment undoubtedly has a beneficial effect on local recurrences. It is therefore feasible to suppose that its effect on the carcinoma cells left after an operation should be equally salutary. In such cases the operation must be as thorough as if no X rays were to be used.

7. Apart from those in the spine, first recurrences after operation in the bones seem very rare, and only two such cases were noted: one in the humerus and the other in the femur.

### CONCLUSIONS.

One is diffident about drawing conclusions from a small series of cases, especially in face of the great difficulty there is in tracing patients for any length of time unless they are persistently written to, as is the practice of so many surgeons to-day. However, one can state without undue dogmatism that certain facts appear to have been elicited.

1. When we find that 28 per cent of all cases survive more than seven years, the operation can be said to have justified itself.

2. The most favourable age for operation is the quinquennium 50 to 54, both as regards duration of life, and also as regards recurrence being delayed for a more lengthy period.

3. In younger patients the prognosis is not so good, and recurrences are more likely to occur early.

4. The absence of deposits of carcinoma in the axillary glands improves the prognosis, and it is possible that the microscopic evidence of chronic inflammation suggests an increased resistance to the disease.

5. Medullary carcinoma is not such a fatal type as it is so frequently considered.

6. Cases showing carcinomatous hyperplasia should be regarded as definitely malignant, and a radical operation performed.

7. Papillary cystic and intra cystic carcinomata are the least malignant, and intraductular carcinoma is only slightly more so.

8. Late recurrences do occur even as long as fifteen years after operation. This possibility almost precludes the establishment of a cure in malignant disease of the breast. It suggests, moreover, that cases should be thoroughly examined at definite intervals, and any recurrence observed should be dealt with preferably as a new focus of malignant disease.

9. Although the length of history given by the patient is misleading, there can be no doubt that the earlier the case is operated on, the more favourable will be the prognosis. All operations must be thorough, and one would be inclined to extend the operation still further by removing the supraclavicular glands in many if not all cases.

10. There appear to be no grounds for believing that the lower inner quadrant of the breast is a dangerous area for carcinoma to originate.

11. A counsel of hope is necessary, in that life is certainly prolonged by the operation. and in many hopeless cases the final stages of the disease are made more bearable for the patient.

In conclusion, I should like to thank the surgeons of the London Hospital for permission to use their cases, and especially would I thank Sir Hugh Rigby, Mr. Sherren, Mr. Hugh Lett, and Mr. Walton for placing their records at my disposal, and also my own chief, Mr. Russell Howard, for much valuable instruction in this subject.

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## URETHRAL POUCHES.

By G. P. B. HUDDY, LONDON.

### INTRODUCTION.

THE earliest reference to urethral pouches appears to be one mentioned in Sir Charles Mansfield Clarke's *Diseases of Females* which was published in the year 1814. The reference, which occurs on page 270, reads as follows: "Sometimes a pouch forms in the posterior part of the urethra, in which a few drops of urine lodge and from which situation it may be pressed out by the finger applied to the part".

Pouches of the male urethra have received scant attention in English medical literature. In 1885 Sir William Arbuthnot Lane<sup>8</sup> described a diverticulum in connection with the prostatic urethra a quarter of an inch in front of the sinus poeularis. Hurry Fenwick<sup>5</sup> recorded a case from the London Hospital in 1886 occurring in a man, age 57. This patient was admitted with urinary extravasation, an incision was made, and eight stones were removed from a pouch situated in the perineum and immediately behind a stricture. He recovered, but later on he returned to the out-patient department, a fresh pouch having appeared. It was situated  $2\frac{1}{2}$  in. from the meatus, and Fenwick suggests that it was due to an anterior stricture, which had been masked by the inflammatory swelling consequent upon the extravasation.

The following article is based largely on the clinical cases and pathological records of the London Hospital. It treats of the condition in the male; pouches in the female are only considered in so far as their etiology bears upon the causation in men.

**Anatomy.**—The male urethra is divided anatomically into the prostatic, membranous, and spongy portions.

The first portion, enclosed within the prostate gland, is  $1\frac{1}{4}$  in. long. Its most marked anatomical feature is the verumontanum or urethral crest, a vertical raised fold composed of mucous membrane and prostatic tissue. The crest is situated on the posterior wall and rises to its maximum elevation in the middle of the prostatic urethra. At its summit is situated the fine slit-like orifice of the uterus masculinus, a small cavity usually about 6 mm. long. On either side of this opening are those of the common ejaculatory ducts, whilst still farther laterally the prostatic ducts open into a depression known as the prostatic sinus.

The membranous urethra,  $\frac{3}{4}$  in. in length, extends between the two layers of the triangular ligament and is surrounded by the compressor urethrae muscle. Situated posteriorly to the membranous urethra on either side of the mid-line lie the two glands of Cowper; their ducts, however, open into the bulb of the urethra and not into the membranous portion. After

piercing the anterior layer of the triangular ligament the urethra enters, and is surrounded by, the corpus spongiosum.

The spongy portion of the urethra is of variable length and opens on the glans penis.

*The Urethral Lacunæ and Glands* are of two varieties: (1) The lacunæ of Morgagni; and (2) The glands of Littre.

1. The lacunæ of Morgagni are found in the cavernous urethra on the dorsal wall and near the mid-line: they are non-secretory, and are lined by the epithelium of the urethra. The lacuna magna is by far the largest of this series, and is situated on the roof of the dilatation of the urethra, just within the meatus, known as the fossa navicularis.

2. The glands of Littre may be divided into those confined to the mucous membrane and a larger type which extends into the submucous layer. They are found in the anterior two-thirds of the cavernous urethra, in dorsal, ventral, and lateral rows.

**Development of the Male Urethra.**—Embryologically the urethra is divided into two portions, the one extending from the bladder to the uterus masculinus, whilst the other comprises the whole of the remaining urethra.

*The Proximal Portion.*—In the early embryonic stages the urinary, genital, and intestinal tracts have a common opening, the cloaca. The partition between the intestine and the allantois grows caudally, so dividing the cloaca into ventral and dorsal segments, the latter subsequently becoming the rectum. The ventral cloaca in time becomes divided, the upper part forming the bladder and the lower the urogenital sinus. The connection between these two segments is a short canal which forms the upper part of the urethra. The urogenital sinus affords a common opening for the two fused Müllerian ducts and this portion of the urethra.

*The Distal Portion.*—At about the fifth week of intra-uterine life the genital eminence appears at the anterior extremity of the urogenital sinus. This genital tubercle elongates to form the penis and a groove appears on its ventral surface; the folds bounding this groove are named the genital folds. The genital folds meet and fuse, thus converting the urogenital sinus and the groove into a canal which is the distal portion of the urethra. The small portion of the urethra within the glans is formed separately from a depression on its under aspect, packed with a plug of ectodermal cells, which later breaks down and so the urethra is completed.

An interesting observation is that made by Johanson<sup>6</sup> on an embryo of 88 mm. In addition to the developing urethral glands "are seen a number of cystic epithelial ducts, some of which are in no way connected with the epithelium of the urethra". They are situated in the connective tissue of the median raphe and have the same epithelial lining as the urethra. Along the mid-line of the rectal wall of the urethra is a definite cord-like thickening of the epithelium, and the appearance suggests that it is being cut off from the urethra. The ducts, mentioned above, have probably been cut off in like manner at an earlier stage.

The uterus masculinus is the fused lower portions of the two Müllerian ducts.

**Types of Pouches.**—The most usual classification is that introduced by Watts<sup>10</sup>:—

1. CONGENITAL.

2. ACQUIRED:—

a. From dilatation of the urethra: (i) Calculus; (ii) Stricture.

b. From perforation of the urethra: (i) Injuries; (ii) Rupture of abscesses; (iii) Rupture of cysts.

The size varies enormously—from the smallest dilatation of the normal glands to a pouch the size of a cocoa-nut, as in Mr. Russell Howard's case reported below. A pouch which gives rise to symptoms is usually about as large as a walnut. The lining mucosa is smooth, and continuous with that of

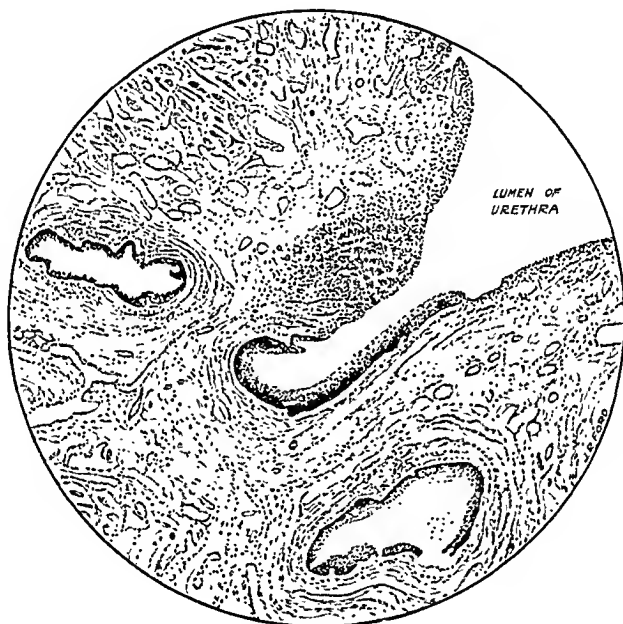


FIG. 33.—Section showing urethral wall with dilated crypts behind a stricture.

the urethra. The aperture of communication may be small or, on the contrary, the pouch may appear as a dilatation of the urethral floor. Infection and inflammation may occur in a pouch in which urine stagnates, and stone formation may ensue.

**Frequency.**—I. R. Sisk,<sup>9</sup> in reporting a recent case, stated that not more than eighty complete cases of urethral diverticula have been recorded. It would seem, however, that this exaggerates considerably the rarity of the condition, for a number of authors have recorded small series, e.g., Bumpus,<sup>2</sup> 4 cases; Escat,<sup>4</sup> 4 cases; and Englander,<sup>3</sup> 2 cases. At the London Hospital also 4 cases have come under observation clinically within the last eight years. In addition, 4 further cases have been found in the records of the London Hospital Pathological Institute during the past ten years.

Urethral pouches are undoubtedly often overlooked because: (1) The symptoms may be very few and unimportant; (2) The signs are often obscure even should a patient present himself for examination. Minute pouches of no clinical significance are frequently met in post-mortem examinations, and extend from the dilated urethra behind a stricture. The condition is illustrated by *Figs. 33 and 34*, which show the appearances both microscopically and macroscopically. Sir Charles Bell,<sup>1</sup> in describing the appearances behind a stricture, stated: "But the most frequent appearances are a number of cavities on each side of the verumontanum. These cavities are only enlargements of the natural ducts of the gland (i.e., prostate). They are sometimes so large as to admit the end of the largest bougie".

**Congenital Pouches.**—These are much rarer than the acquired variety. Of sixteen congenital cases analysed by Watts, all except two were found in relation to the penile portion of the spongy urethra. *Case 3*, described in this article, affords an example of a congenital pouch in which the communication with the urethra was noted  $1\frac{1}{2}$  in. in front of the triangular ligament.

Many theories have been advanced to explain the origin of congenital pouches. As the cavity is found on the ventral aspect of the urethra, two theories which could explain the condition are: (1) Imperfect fusion of the two genital folds; and (2) That one of the cystic epithelial ducts described by Johnson may retain its urethral communication and become dilated. Some, however, may arise from the Müllerian ducts, but they must be very rare. Escat<sup>4</sup> recorded a case which he regarded as of this type, in which a diverticulum "the size of a nut", situated at the posterior part of the scrotum, communicated by a long canal with the posterior urethra.

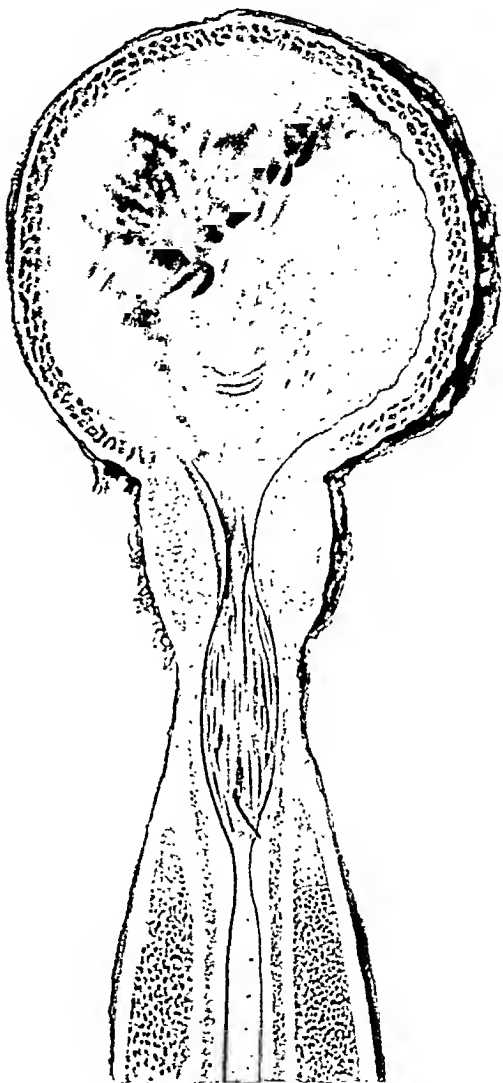


FIG. 34.—Specimen showing urethra and bladder. There is a stricture of the bulbous urethra, proximal to which are several dilated crypts, in one of which a bristle has been placed.

An extremely interesting specimen in the London Hospital Pathological Institute (*Fig. 35*) illustrates the possibility of this mode of origin. The specimen was obtained from a male, age 51, who had died from hæmorrhage after a gastrojejunostomy. There was slight hypertrophy of the bladder. The urethra shows a cystic dilatation of the uterus masculinus 1.5 by 1.2 cm. Microscopic sections have been taken, and Professor H. M. Turnbull reports as follows:—

In microscopic sections the cavity contains a yellow coagulum, and is lined by a single row of low cubical or flat epithelial cells. The lining rests upon a definite wall, which is composed of circularly directed muscle-fibres and contrasts conspicuously with the prostatic stroma in its abundance of stout elastic fibres and its relative scarcity of collagenous fibres. Within this wall are a few acini, which are lined by similar epithelium and which are usually filled with a similar coagulum.

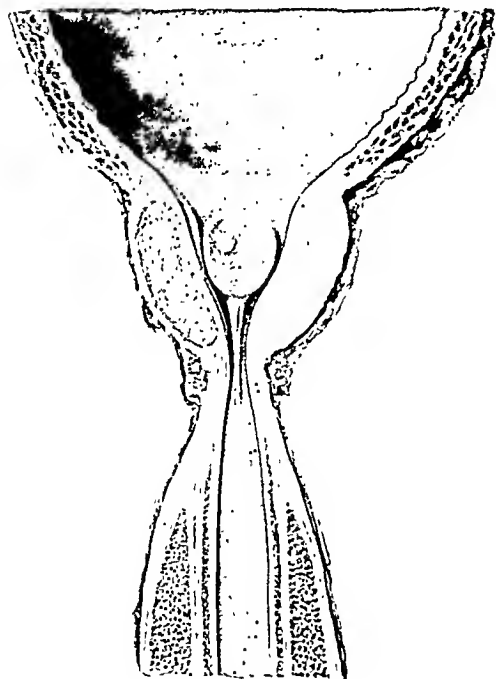


FIG. 35.—Specimen showing dilatation of the uterus masculinus.

### Acquired Pouches.—

**RELATION TO URETHRAL OBSTRUCTION WITH DILATATION OF THE URETHRAL GLANDS.**—That the glands and lacunæ are frequently slightly dilated behind a stricture has already been mentioned and illustrated by diagrams. A rather more exaggerated example has been noted in the pathological records.

*Necropsy 181/1914.*—A tailor, age 16, died of cerebral hæmorrhage complicating cardiovascular hypertrophy and nephritis. The kidneys were hydronephrotic and showed focal areas of fibrosis with cyst formation. The bladder and ureters were dilated. There was a diverticulum of the penile urethra and a large scar surrounding and constricting the meatus. Clearly if this condition were progressive it would

result in the formation of pouches not only of pathological but also of clinical interest. Nevertheless this is probably an unusual method of formation of large pouches, for although the glands are found on all walls of the urethra, diverticula are found on the ventral wall only.

Johnson<sup>7</sup> has recorded a case of a large scrotal cyst with a pedicle which could be traced to a point corresponding in position to the duct of the left Cowper gland, thus indicating the possibility of a pouch being developed from these glands.

**RELATION TO URETHRAL OBSTRUCTION WITH PERI-URETHRAL ABSCESS.**—An entirely different mode of formation in association with stricture is illustrated by the following case:—

**Case 1.**—A traveller, age 34, admitted in 1924, under Mr. E. C. Lindsay, with acute retention of urine.

**HISTORY.**—The patient had suffered from gonorrhœa in 1912, and this was followed by dysuria two years later. In 1919, when at sea, he experienced an attack of acute retention of urine, which was relieved naturally by a hot bath. On the following evening retention again occurred, when a hot bath failed to give relief. As catheterization by the ship's doctor failed, the patient was taken to a general hospital ashore. Under a general anæsthetic a No. 4 metal catheter was passed and tied in for a very prolonged period. The formation of an abscess ensued,

at a point about  $2\frac{1}{2}$  in. from the external meatus, and at the site of the present sac. This was accompanied by rigors and pyrexia. Pus was squeezed out, and later the catheter was removed. Dilatation was carried out with an expanding bougie, and the patient was discharged from hospital.

In 1921—and again at sea—retention recurred.

Hot baths and catheterization by the ship's doctor failed, partial relief eventually being obtained by the passage, in small quantities and with much

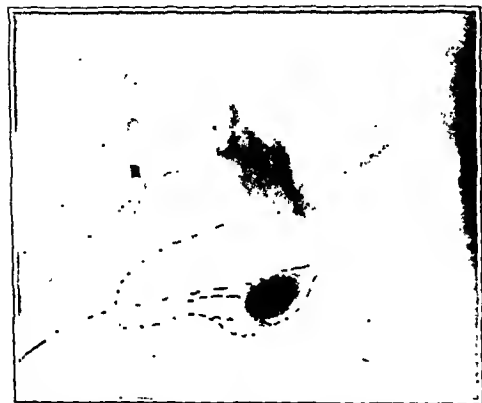


FIG. 36.—Radiogram showing acquired urethral pouch distended with sodium bromide. (M. H. Jupe.)

pain, of ten ounces of urine. At the British Hospital, Port Said, dilatation was carried out in two stages until a 13/15 Lister bougie could be passed. During the ensuing years the stream on micturition gradually lessened. A month before admission the dysuria was so marked that a small sound was passed. Spasm and retention ensued and were again relieved by instrumentation—this occurred on three occasions.

He was admitted to the London Hospital suffering from urinary retention, and was relieved by suprapubic puncture of the bladder. Five days later an internal urethrotomy was performed and two strictures were discovered, one  $2\frac{1}{2}$  in. from the meatus and one in the bulb. A urethral pouch was discovered, situated at the penoserotral junction, and was, when distended, the size of a walnut. He was transferred to, and is still attending, the Stricture Section of the Genito-urinary Department, where an X-ray picture of the pouch distended with sodium bromide was taken, and is shown in Fig. 36. The condition as seen at present is illustrated by Fig. 37. On urethroscopic examination the communication with the urethra is wide and the cavity projects down from the urethral floor. The wall is smooth and of a white colour.

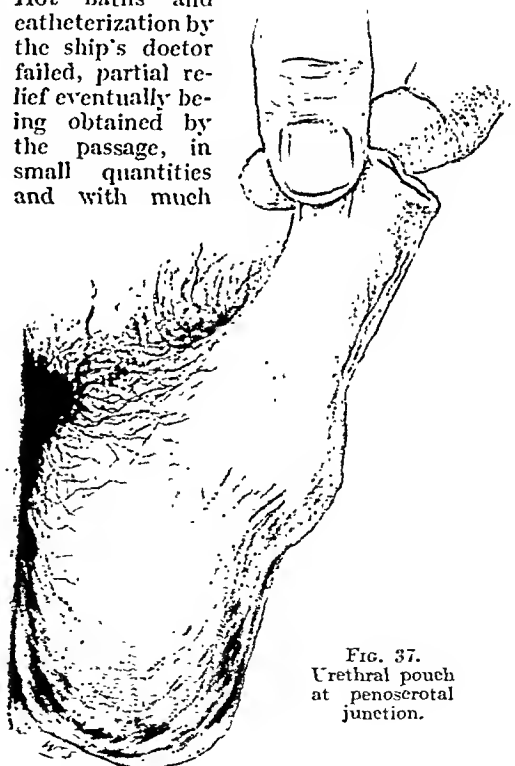


FIG. 37.  
Urethral pouch  
at penoserotral  
junction.

**Urethral Pouches in Women.**—This condition in the female will be considered only in so far as it has a bearing on the causation of the same pathological condition in the male.

The female urethra corresponds to that part of the male urethra which extends from the bladder to the uterus masculinus, a very uncommon site for pouches. As urethral stricture is also very rare and does not appear to be associated, it seems certain that there must be some additional factor operating in the female to account for diverticula. The physiological process of child-bearing at once suggests itself as the possible factor, and this is supported by the clinical features of recorded cases. The usual history is that the swelling followed a confinement.

During delivery the urethra becomes very much elongated and is subjected to pressure. As the urethra has an anterior concavity, the convex vaginal wall will be more liable to damage than the pubic wall. A urethro-vaginal fistula may be thus produced. If, however, the injury is of a lesser degree, the damage to the urethral wall may result in the formation of a hernial protrusion—in other words, a pouch.

*Case 2.*—A multipara was admitted to the V.D. (Female) Department, in February, 1923, under Mr. Hugh Lett. On examination a pouch which contained half a drachm of fluid was found. The opening of the diverticulum was seen with the urethroscope to be situated in the posterior urethral wall.

**Injury as a Factor.**—This follows immediately on the preceding paragraph, for as trauma is probably a factor in the female, so it may be in the male. Watts and Sisk have reported cases of acquired diverticula following urethral rupture. Operations and false passages may also be followed by the formation of diverticula, an example being obtained from the Pathological Institute.

*Neeropsy 116/1921.*—Male, age 66. Died following an operation for duodenal ulcer. The mid-penile urethra showed scarring. Posteriorly, in the region of the bulb, was found a smooth, lined pouch, 1 cm. in depth; it was directed downward, and was adherent to a linear scar in the skin of the perineum. The explanation of the formation of this pouch is that an external urethrotomy had been performed behind the stricture. The obstruction to micturition consequent on the stricture was not of such a degree as to result in fistula formation, and the skin wound closed. The deeper portion of the wound was distended each time the bladder emptied, and so failed to unite, and in due course became covered with a layer of epithelium.

**Symptoms and Signs.**—Clinically the cases are of two types:—

1. A patient presents himself with rather vague and indefinite symptoms, and a diagnosis can only be made by means of the urethroscope.
2. He complains actually of a swelling at some point along the urethra. These will be illustrated by the following examples.

**Type 1.**—*Case 3.*—A railway clerk, age 22, was admitted to the London Hospital, in 1922, under Mr. Hugh Lett. He had suffered from no previous urethral disease.

**HISTORY.**—His complaint was that for three years, although micturition was free and normal, it was followed by an annoying dribbling of urine in drops, which caused him most inconvenience during the winter. During the preceding year the trouble had been more marked and accompanied by a slight degree of increased frequency of micturition.

**EXAMINATION.**—Diagnosis was made by the urethroscope, when a pouch was

seen on the floor  $1\frac{1}{2}$  in. in front of the triangular ligament. It extended posteriorly and just below the urethra, the wall separating the two being thin, for the air-distended pouch ballooned into the lumen of the urethra.

Fig. 38 shows an X-ray picture of this pouch distended with sodium bromide.

**TYPE 2.**—*Case 4.*—A pensioner, age 63, was admitted to the London Hospital in 1916 under Mr. Russell Howard. He complained of a swelling in the perineum.

**HISTORY.**—Two years previously he had noticed a swelling, which increased until it attained the size of a hen's egg. It then burst and discharged clear fluid. This sequence of events had recurred, and a similar swelling had now appeared for the third time. It was not tender, but there was a burning pain at the site after micturition. During the week preceding admission there was considerable difficulty in micturition and the swelling increased enormously in size.

**EXAMINATION.**—Situated in the perineum was a cyst the size of a large cocoa-nut which extended into the perineum, pushing the testes before it. The temperature and pulse were normal.

**OPERATION.**—The patient was placed in the lithotomy position. A mid-line perineal incision was made and deepened until a smooth-walled cavity was opened. One pint of clear urine was evacuated. On further examination the cavity was found to communicate with the urethra; the communicating orifice was cauterized and sutured. The serotal tissues were united with catgut to obliterate the cavity, and the perineal wound was closed.

I have endeavoured to trace this patient, but without success.

The symptoms may be modified by the onset of complications, such as inflammation giving rise to increased frequency and painful micturition, with a discharge of purulent urine when the pouch empties. The presence of stones has been already noted; an example was found amongst the pathological records. A male, age 49, was admitted with and died of hyperpiesis. During the routine pathological examination brown calculi, up to 0.3 cm. diameter, were found in the dilated urethral crypts. (*Necropsy* 233/1921.)

It is with pleasure I record my thanks to Mr. Hugh Lett, the Director of the Genito-urinary Department, for his help in the preparation of this article, and to Professor H. M. Turnbull for advice concerning the pathological specimens. Mr. Russell Howard and Mr. E. C. Lindsay have very kindly permitted me to use their cases.



FIG. 38.—Radiogram showing congenital pouch distended with sodium bromide. (M. H. Jupe)

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## THE TECHNIQUE OF EXTRAPLEURAL THORACOPLASTY.\*

BY H. MORRISTON DAVIES, RUTHIN, NORTH WALES.

### HISTORICAL.

THE birth, growth, and development of an idea or project is always a matter of great interest. For the surgeon, the evolution of a new type of operation or a new method of attack on a disease has a very special appeal. This is particularly so when the earlier efforts give promise of success, and the process of evolution can be followed until it reaches the stage of simplification and exactitude of the essential principles, at which, for a time, it remains comparatively stationary. During the last three and a half decades the operation of extrapleural thoracoplasty has passed through its period of immaturity and has reached that of adolescence, with a fine record of success that augurs well for its future and for its more extended use.

The credit for the conception of the idea belongs to Quincke and to Carl Spengler. They recognized the mechanical difficulties associated with the healing of the lung, and especially of cavities therein; and that to overcome these difficulties mobilization of the chest wall was necessary. To this end they, in 1888 and 1890 respectively, advocated multiple small rib resections. Spengler gave to this principle the name 'extrapleurale thoracoplastie'. Turban, in 1899, carried this idea somewhat further, resecting portions (totaling 65 cm.) from six consecutive ribs. The first decade was therefore a period of very slow development.

During the earlier years of the twentieth century no further advance was made, though Garré and Quincke attempted a modified thoracoplasty for hæmoptysis. In 1907, Friedrick, operating for Brauer, did the first operation which has become known by their joint names. This consisted of a decostalization of the side of the chest. It was extensively practised for a time both by Brauer and Friedrick and by Sauerbruch. The operation had a very high mortality, because the removal of all rigidity from one side of the thorax gave rise to paradoxical movements of that side and to a to-and-fro flapping of the mediastinum. The effect of these is deficient ventilation of the lung, imperfect aeration of the blood, and serious disturbance of the heart which may lead to heart failure.

Wilms, recognizing that the dangers of the Brauer-Friedrick operation were due to the loss of support caused by the complete removal of the bony framework of the chest, devised (1911) the operation which goes by his name. He resected 2 to 4 cm. of the posterior parts of the first eight ribs through a paravertebral incision, and, later, the costal cartilages of the first five through

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\* Partial thoracoplasty, in conjunction with or independent of artificial pneumothorax, is not under consideration.

a parasternal incision. The upper ribs, being thus detached, back and front, were free to drop down and inwards and to become approximated to the mid-line. The collapse so produced, though far from complete, proved most efficacious.

It is, however, to the acumen of Sauerbruch that we owe the operation as it is almost universally done to-day by those who practise thoracoplasty. He recognized that the all-essential factor in the production of unilateral collapse of the one side of the chest wall is the excision of a portion of almost all the ribs on that side, always up to and including the first, *as close* to the transverse process of the vertebrae as possible. When this is done, no operation on the front of the chest is, as a rule, necessary. This operation is known as 'Sauerbruch's paravertebral extrapleural thoracoplasty'. Brauer and Friedrick have abandoned their original method, and they now obtain collapse by an operation very similar to that of Sauerbruch. The only essential difference lies in the amount of bone removed.

Whilst there is agreement among the surgeons on the general principles of the operation, there are several differences of opinion on the individual details of the technique. These it is proposed to indicate.

### TECHNIQUE.

**Preliminaries.**—The patient should be kept quiet in bed for ten days before the operation. It is most essential that there should be no recent complicating feature, such as a coryza, however mild, or diarrhoea, etc. Every effort must be made to have the lungs as free from secretion as possible. This is done by expectorants, and by posture when such is of assistance in the individual case. It will as a rule be many hours after the operation before the patient is able to expectorate; therefore the clearer the lungs are before the start, the less the danger.

The author gives his patients a course of tablets of suprarenal extract for ten days, but it is quite an open question if this has any real value. The rest of the preparation is the same as for any other operation. Brunner recommends a course of bromides; J. Alexander refers to the use of digitalis if the heart is weak.

**Anæsthetization.**—The fear of pulmonary complications and of further damage to an already diseased lung has resulted in establishing a considerable degree of antagonism to the use of general anæsthetics. A few years ago, most of the operations of thoracoplasty were done entirely under local analgesia. At the present time there are still some (*viz.*, Brauer and Wendel) who maintain that the local method of anæsthesia is the only permissible one. Most surgeons, however, are beginning to realize that the danger of shock resulting from the intense strain of consciousness during the operation may be as serious as the risks of a general anæsthetic. Holding, personally, very strong views on this, it is gratifying to find so great an authority as Brunner making the following statement: "General anæsthesia has undoubtedly great advantages over local. The psychical shock of the operation, which is a very important feature in operations done without loss of consciousness, is greatly diminished by general anæsthesia. We have repeatedly convinced

ourselves that the general condition of the patient is unquestionably better after general than after local anæsthesia. The difference shows itself above all in the condition of the pulse."

A few surgeons, among them Archibald, Gravesen, and the author, use a combination of local and general anæsthesia. The majority use local anæsthesia when there is much sputum (Sauerbruch, if over 30 c.c. per diem; Brunner, if over 20 c.c.; Bérard, if over 60 to 80 c.c. or the patient is dyspnœic). If there is but little sputum they use ether.

The local anæsthetic used is novocain with adrenalin hydrochloride; but Bérard prefers anacain, which renders the tissues anæsthetic for thirty-six to forty-eight hours. It must be remembered that novocain is a poison, and that occasionally patients show an idiosyncrasy to it. It was for this reason that Saugman used to test the effect of it on his patients beforehand. The injection of 150 c.c. of a 0.5 per cent solution is regarded by Sauerbruch as safe; he does not exceed 200 c.c. Some surgeons, however, such as Lorey and Bull, use double the quantity. The combination of morphia with novocain increases the dangers, and for this reason Sauerbruch and Jessen limit also the morphia to  $\frac{1}{4}$  gr. The author finds that by the use of general anæsthesia with the local considerably less of the latter is necessary, and now never uses more than 60 c.c. of a 1 per cent solution.

When a general anæsthetic only is given, deep anæsthesia is required, according to Brunner, to abolish the reflexes. Ether is, as a rule, preferred, but sometimes nitrous oxide and oxygen are used. Brunner quotes Spengler's investigations in 1903, which showed that ether is less deleterious for phthisical patients than chloroform. This is not altogether the experience of the author, who, while recognizing fully the dangers of both anæsthetics, prefers chloroform for the very light anæsthesia required, supplemental to the local, in order to keep the patient asleep during the operation.

The question of post-operative pain is of great importance, as on the absence of this depends so much the ability and the willingness of the patient to cough and to bring up the accumulated expectoration. It is for this reason that the author injects into the neighbourhood of each nerve 5 min. of absolute alcohol, so as to obtain an anæsthesia which will continue until the healing processes—including the rounding off of the cut ends of the ribs—have become complete. Nyström crushes the nerves and injects alcohol into them. Others, including Jessen and Schreiber, also Sauerbruch in those cases which have a very thickened pleura, resect from 1 to 2 cm. of the nerve. Experience has shown that paralysis of the lower intercostal nerves produces only very rarely serious consequences on the abdominal wall; but Brauer, because of the possibility of paralysis, insists that the nerves shall be preserved. Oehlecker has shown that, after nerve resections in operations for empyema, the paralysis may lead to a ventral hernia.

When the operation is done under local anæsthesia entirely, the Schumacher technique is undoubtedly the best.

**The Number of Stages.**—This is another somewhat vexed question, and opinions differ considerably. Sauerbruch does the operation in two stages, but occasionally in one, three, or even four. Nyström used to do it in one, but now invariably operates in two stages. Bull and Jacobæus and Key

believe in the two-stage operation; Wendel and Gravesen in the one-stage. Jehn does it in one stage if the heart is strong and the mediastinum is fixed. Undoubtedly much must depend on the condition of the patient and the character of the disease. When the operation is done in two stages, ribs 11 or 10 to 5 inclusive are removed at the first stage, and ribs 4 to 1 at the second.

The effect on the mind due to the strain of anticipation and of the actual operations themselves must unquestionably be infinitely greater if there are two major operations as compared with one, especially if both are done under local anæsthesia. The patient has scarcely recovered from the shock of the first when he has to endure that of the second. This is particularly the case since, in the two-stage operation, the sooner the second stage is done after the first, the better will be the results so far as collapse is concerned. Too long a wait after the first operation will allow the re-formation of bone from the periosteum which has been stripped off and left behind, and the success of the operation becomes jeopardized. Nyström, to overcome this difficulty, removes the periosteum with the ribs. The interval between the two stages is usually two or three weeks. Another objection to the two-stage operation is raised by Gravesen, who considers that the second interferes with the satisfactory healing. Jacobaeus and Key, however, do not agree with this.

Against the one-stage operation is the increased risk due to the greater magnitude of the undertaking; the longer time taken, and the greater wound exposure; the greater and more sudden collapse of the lung; and the risk of more serious interference with the mediastinum and heart.

J. Alexander records the advantages of the two-stage operation as: (1) Less shock and less sudden changes demanded of the circulatory and respiratory mechanisms, which are enabled to adjust themselves gradually. (2) Fewer toxic products are pressed into the general circulation to endanger latent lesions in the better lung; therefore two stages are important if the better lung is under suspicion of activity. (3) The first stage usually causes enough rest and relaxation of the lesions to bring about a definite clinical improvement.

He states the advantages of the one-stage operation to be: (1) Much preferred by the patient. (2) Risks of embolus and of anæsthetic poisoning and of wound infection run once only. (3) If wound infection should occur, the whole lung has been compressed—no need to postpone a final stage of the operation. (4) Muscles are sectioned once only. (5) More even lung compression and so less irritation of the cavities and fewer chances of lung hæmorrhage. The scapula fits more evenly against the lung (Gravesen).

Brunner states that the one-stage operation is preferable, not only as regards operative technique, but also from the mechanical standpoint. He emphasizes, however, that it should be done only in fever-free cases with slightly progressive or stationary fibrotic tuberculosis with small cavities and little sputum; that the general condition must be good and the circulatory system sound.

Baer considers the one-stage operation as the ideal; but contra-indications to it are, imperfect closure of the glottis on coughing, and excessive displacement of the mediastinum associated with maximum compensatory emphysema of the opposite lung. Hanke states that thoracoplasty, whether done in two

or more stages, is not an operation for a patient seriously ill. If the mediastinum is not sufficiently fixed, paradoxical respiration with great disturbance of breathing and interference with expectoration must be expected after the one-stage operation.

Much depends on the condition of the patient and on the character of the lung lesion. Sauerbruch and Brunner operate in one stage on those patients only who have a very chronic fibrotic lesion. The author is not so precise in his limitations. His attitude is as follows: The operation should be done in one stage if possible, but always having the greatest regard to the safety of the patient. If the patient's general condition is good, if there is no difficulty in respiration when on the table, and the colour is satisfactory, if the sputum is not excessive and likely to flood the bronchial tubes when the lung collapses, and the disease is not acutely active with considerable complicating secondary infection, then the operation is started off with the complete incision as for a one-stage operation. If there is any doubt, however, as to the patient's ability to tolerate the complete rib resection, the operation is begun as though for a two-stage undertaking. When this first part is completed up to and including the rib excision, the condition of the patient is considered, and if this appears to justify it, the second stage is carried out.

**The Skin Incision.**—The Sauerbruch paravertebral incision for the removal of parts of ribs 11 to 1 in one stage, starts above, just below the upper edge of the trapezius muscle, extends down between the vertebral border of the scapula and the spines of the dorsal vertebræ (the arm being held close to the side) at a distance of  $3\frac{1}{2}$  or 4 in. from the spines. When the incision reaches the 11th rib (or the 10th if ten only are being removed), the incision curves abruptly outwards to the posterior axillary line. When the operation is done in two stages, for the first, the lower part of the incision is used, starting above over the 4th intercostal space. At the second stage the upper part of the incision is modified by being curved outwards along the 4th rib.

Brauer amplifies the vertical incision by a transverse one along the 1st or 2nd rib. This he regards as especially useful when doing the second stage in a two-stage operation. Wendel, operating in one stage, starts his incision over the 4th rib. To reach the upper four ribs he tunnels under the trapezius. He claims that this is quicker, requires less anæsthetic, and prevents such free division of the vascular muscle.

**The Number of Ribs Resected.**—Portions of the first ten ribs are almost invariably resected. The one exception is the procedure of Brauer, who occasionally leaves the first rib undivided when there is much retraction of the apex of the lung. The importance of freeing this rib is, however, great, because as each rib is slung on to the one above by the intercostal muscles, so are they all eventually slung on to the first. After resection of portions of the first ten ribs, the compression of the lung is dependent on the alteration of the position of the ribs in three directions: (1) The cut ends are approximated; that is to say, the cut freed end of the rib approaches to the spine. (2) The cut freed ends of the ribs drop down a distance equal to one, and often two vertebræ. (3) There is a bucket-handle action which tilts the main part of the rib through part of an arc of a circle, bringing the centre of

the curve of the rib nearer to the middle line and at a lower level. This change, which was a very striking one after the Wilms operation, is still present in the paravertebral resection by reason of the yielding of the costal cartilages to torsion. Both the second and the third of the above-described movements are limited if the first rib remains intact.

Saugman did not excise any of the 11th rib. Sauerbruch, Brunner, and most of the other surgeons do. They claim that by removing some of the bony support of the diaphragm that muscle is relaxed, and, rising into the chest, increases the collapse of the base of the lung. The author does not agree that division of the 11th rib is necessary. Ample collapse can be obtained without this; it is at the apex rather than at the base that the collapse does not always fulfil all requirements. Moreover, the retention of some of the supports of the diaphragm is advantageous, in that this muscle exercises a steady effect on the lower part of the mediastinum, including the heart.

Resection of the ribs is almost always done from below upwards (except occasionally only when the disease is almost entirely basal). This order of resection enables the lower part of the lung to collapse first, and so prevents aspiration of infected secretions from the upper into the lower lobes.

**The Amount Resected.**—All are agreed that the part of the rib resected must extend up to the transverse processes of the vertebræ. The actual length of rib excised is of less importance, except that if too much is removed there is the risk of those dangers associated with the original Brauer-Friedrick operation (*viz.*, mediastinal fluttering and heart failure). On an average 2 to 4 cm. only is removed from the 1st rib, 6 to 8 cm. from the next three, from 10 to 16 cm. from the next four, and then again less from the lower two or three. The total amount excised by Sauerbruch averages 110 cm. Jahn and Brunner excise somewhat less; Brauer and Friedrich average 125 cm.; the author 120 to 130 cm.; Jacobæus and Key 140 cm.; and Bull from 120 to 180 cm. Brauer maintains that all that part of the ribs underlying the scapula should be removed. It is most important for efficient collapse that, after the operation, the scapula should sink right in so that it lies at a deeper level (*i.e.*, more anterior) than the posterior surface of the transverse processes. Owing to the changes in position of the freed end of the ribs, already mentioned, this falling in will take place even if the whole of the ribs underlying the scapula are not excised.

#### DETAILS OF THE AUTHOR'S TECHNIQUE.

**Anæsthetization.**—Previous to the start the patient must have been encouraged to cough up all the sputum possible. Omnopon ( $\frac{1}{3}$  gr.) is given hypodermically twenty minutes before the local anæsthetization is begun. This anæsthetization is done in bed an hour before the operation. It is considered inadvisable to have the patient in the theatre and on the table all this extra time, as it increases the period of anticipation, and the operating table is uncomfortable, especially for a patient who is at all emaciated.

The anæsthetic used is novocain (1 per cent) to which has been added adrenalin hydrochloride (1-1000) 12 drops to 60 c.c. of the solution. The

line of the skin incision  $3\frac{1}{2}$  in. from the mid-line is first anæsthetized, starting over the 10th rib and working upwards to beyond the 1st rib; then from the starting-point along the course of the 10th rib to the posterior axillary line. Two syringes are then used: the one of 2 c.c. capacity and the other of 1 c.c. The smaller is filled with absolute alcohol, and the larger with novocain. The latter is fitted with its needle (very fine and long), which is driven through the anæsthetized skin inwards and backwards into the 10th interspace, a little novocain being injected all the time. The rib is sought for and touched so as to locate it and gauge its depth; when found, the needle is then redirected into the intercostal space below, and the rest of the novocain injected between the two ribs. The syringe is detached from the needle, which is left *in situ*, the 1-c.c. syringe is attached to it, and 5 min. of absolute alcohol are injected into the interspace, the needle being slowly withdrawn a few millimetres as the alcohol runs in. The 1-c.c. syringe is in turn detached, the needle being still left in the tissues for the time being to mark the space treated. Another needle is fitted to the 2-c.c. syringe, refilled with novocain, and the process is repeated in the space above. When this space has been anæsthetized and the syringe detached from the needle, the first needle is withdrawn for use for the injection of the next space above. This process is repeated until the 1st intercostal space has been anæsthetized. Novocain only is injected above the 1st rib. Finally, a few syringe-fuls of novocain are injected into the subcutaneous tissues and the muscles along the lines of the incision.

Just before the scheduled time of the operation, the patient is taken to the theatre and arranged on the table. He reclines on the sound side with the shoulders raised and the pelvis and buttocks well supported. A nurse steadies the arm on the affected side. Morphia ( $\frac{1}{8}$  gr.) and atropine ( $\frac{1}{120}$  gr.) are given.

The general anæsthetic is not started until the surgeon is absolutely ready, so that he can begin the moment the patient is sufficiently under. As the tissues have all been anæsthetized it is not necessary, nor is it desirable, to get the patient deeply under. The lightest anæsthesia only is requisite. As stated above, the author prefers chloroform for this light narcosis.

**Operation.**—Sauerbruch's paravertebral incision is used. If the general condition is good, if the cardiac muscle has not been seriously impaired by toxæmia, if the colour is satisfactory and the respirations under the anæsthetic are free and 'comfortable'; if, that is to say, there is every indication that the patient will tolerate well the complete operation in one stage, then the incision is begun above the 1st rib, carried down to the 10th, and then outwards to the posterior axillary line. The skin and the muscles are divided down to the ribs. The muscles are: the trapezius above and the latissimus dorsi below; deep to the trapezius is the rhomboideus major, beneath which again is the serratus posticus superior; below the rhomboideus is the vertebral aponeurosis, and below this again some of the fibres of insertion of the serratus posticus inferior will be cut. The division and separation of these muscles exposes the longitudinal fibres of the accessory; the outer border of this muscle is defined, the outer fibres are detached from the ribs and displaced inwards. The posterior parts of the ribs and intercostal muscles are now exposed.

It is most important to ensure that all bleeding points have been secured;

all artery forceps should be replaced by ligatures, as the forceps are apt to be dislodged during the subsequent movements necessary for stripping and excising the ribs. The skin and the muscles should be protected as completely as possible by gauze secured to the wound.

The next step is the stripping of the periosteum from that part of the ribs which it is proposed to excise. In order to get a fuller exposure of the field of operation, the nurse draws the arm on the affected side forwards and upwards towards the head; this carries with it the scapula and the attached muscles. The periosteum is incised with a knife along the dorsum of the rib; it is then stripped off the dorsum by short vigorous strokes with a square-ended periosteal elevator; just beyond the angle the periosteum is detached from the upper and lower borders of the rib as well. A Doyen elevator (either the ordinary one, or one with the elevator set at a more obtuse angle to the shaft) is slipped round the rib between it and the periosteum, and the bone is stripped back to the transverse process. For the separation of the periosteum and intercostal muscles from the anterior part, the author uses his own elevator (*Fig. 39*), which is easily placed in position between the bone and periosteum in front of the angle, and can then be driven forwards, out of sight under the muscles if necessary, without risk of damage to any other tissues. The 10th to the 5th ribs inclusive are thus treated, and in this order. As each rib is freed, the lung and soft tissues retract away from it, leaving a space which must be gently packed with gauze rung out in hot saline solution.

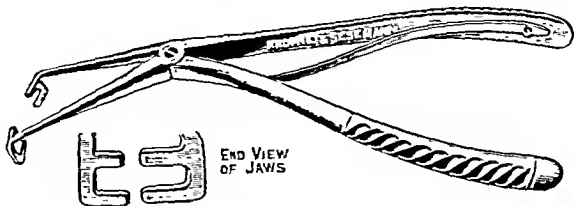


FIG. 39.—Author's raspatory.

The desired length of each of these ribs is now excised, starting again with the 10th. Each rib in turn is cut through with stout long-handled bone forceps as close to the transverse process as possible. The rib, as it is cut, is seized by the assistant with lion forceps, and the cut end lifted out of the wound. The ring of the author's bone forceps is slipped on to the rib and is run along it as far forwards as the periosteum has been stripped (i.e., until it meets the resistance of the unseparated intercostal muscles); at this point the bone is divided and removed. These forceps (*Fig. 40*) ensure that the cutting jaws of the forceps stay in contact with the rib; they allow of the division of the bone with absolute safety, out of sight if necessary.

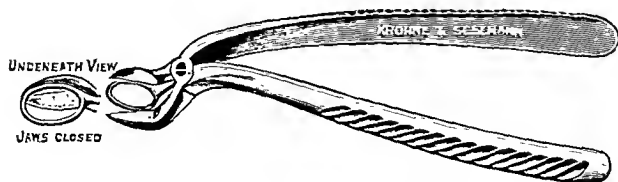


FIG. 40.—Author's bone forceps.

The condition of the patient is reported on at this stage of the operation.



If everything is satisfactory, the excision of the required amount of the upper four ribs can be proceeded with. The 4th and 3rd ribs are dealt with in the same way as the lower ones. The 2nd is then excised; for the separation of the periosteum of this rib, Doyen's raspatory only is used. The portion of bone having been removed, access to the 1st rib is easier. As clear a view as possible must be obtained. The posterior edge is cleaned, then the upper surface, and then the lower. The blunt point of a Friedrick's raspatory is now passed round the inner margin—all the time keeping in closest contact to the bone—and the periosteum is freed over a distance of about  $2\frac{1}{2}$  cm. The raspatory is withdrawn, and a narrow copper spatula, with one end curved back as a hook, is passed along the under surface and round the inner edge. This spatula protects all adjacent tissues from injury while the rib is cut with blunt-point forceps. As soon as the 1st rib has been cut, no more anæsthetic is given.

The vertebral end of each rib is examined and, if necessary, pointed or rough particles are cut away with gouge forceps. All loose tags of muscle are cut away, and all bleeding points ligatured. The muscles are then accurately sutured in two layers with catgut, and the skin incision is closed, except at the lowest point, through which a large drainage tube is inserted extending upwards some four inches under the superficial muscles.

The dressings are applied and fixed with a strong broad many-tailed bandage with shoulder straps. It is of the utmost importance that this bandage shall be applied firmly, so as to give adequate support to the collapsed side of the chest and to the heart, but not so tightly as to compress and restrict the sound side. To obviate this restriction Jacobaeus and Key make, before the operation, a plaster cast of the sound half of the chest; after the operation, they fix on this half-jacket and apply the bandage over the dressings on the side of the operation and over the plaster jacket on the other side. The author has not found this to be necessary. The arm must be included in the bandage. So little anæsthetic should have been given that the patient is recovering consciousness before leaving the theatre, and is aware of his surroundings by the time he is settled back in bed.

The amount of rib removed is from 120 to 130 cm. This total is made up as follows: 1st rib, 4 cm.; 2nd rib, 8 cm.; 3rd rib, 12 to 13 cm.; 4th to 8th ribs, 15 to 17.5 cm.; 9th rib, 12 to 13.5 cm.; 10th rib, 9 to 10 cm. The time taken by the author for the complete one-stage operation is forty minutes. Sauerbruch, with his big experience and special assistants, can do either stage in twelve to twenty minutes. Alexander says: "Jessen can do the complete operation, ribs 11 to 1, in twenty minutes. Bull's average time, with one or two assistants, is one hour. Either stage of the operation can be performed handily by a well-trained team in forty minutes".

**After-treatment.**—The patient is propped up in the semi-recumbent posture as soon as the recovery of the cardiovascular tone justifies this. Immediately on his return the patient is given a rectal injection of saline. This is repeated six-hourly until the next morning. Plain water or water containing sodium bicarbonate is allowed. There is usually so little shock that stimulants are not required, and there is usually so little pain that anodynes are not needed. Omnopon ( $\frac{1}{3}$  gr.) is given hypodermically the first

night to ensure comfort and to help sleep. Should pain be severe, morphia ( $\frac{1}{8}$  gr.) is given, and is repeated when necessary. The patient is encouraged to cough after the hypodermic of omnopon has been given, and again the next morning. If it is impossible then owing to the pain, and there is the fear of complications from retained secretions, it is advisable to give another hypodermic so as to ease the pain and enable the patient to bring up the sputum.

It is more than probable that the dressings will be 'through' the first evening; they must not, however, be changed, but must be 'packed' only, i.e., fresh dressings are placed over the bandage, *which must in no way be disturbed*. The first dressing is done twenty-four hours after the operation. Very great care is required to avoid pain when turning the patient, and the serious disturbances which may arise when the support is removed from the collapsed side of the chest. The pain is occasioned when rolling the patient over on to the sound side; the cut ends of the ribs are certain to shift and to irritate the nerves and the pleura. The rotation of the patient should be done entirely by means of the draw-sheet and before the bandage is disturbed. Fresh draw-sheets and everything likely to be required must be at hand, so that the bed can be put in order with the one set of turnings. Arranged on a towel at hand must be the fresh set of dressings—gauze and wool complete—ready to be picked up and placed on the wound. While the patient is turned on to the sound side, the arm is supported by a nurse so as to avoid all drag on the divided muscles. The bandage is undone and removed with the dressings. The tube is taken out, the wound is lightly swabbed, the prepared dressings are applied and held in place while the draw-sheet and the one set of tails of the bandage are tucked under the sound side. The patient is now rolled back by the old draw-sheet, while being supported on the other side by the clean one, and the bandage is applied as quickly as possible. The arm is this time secured by a separate sling and supported under the elbow by a pillow. The bandage should not be disturbed again until the stitches are taken out.

Once the wound is soundly healed, the many-tailed bandage can be dispensed with. Support for the collapsed side is, however, very essential for the next few months, until such time, at any rate, as the chest wall has become fixed by the new bone formation, which replaces the ribs removed and at the same time ankyloses the adjacent bones to each other. This support should be in the form of a pad, pressing especially on the axillary surface and over

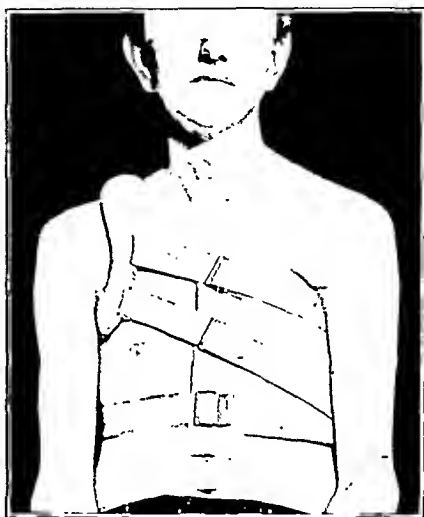


FIG. 41.—Support used by author after thoracoplasty.

the scapula on the side of the operation; the straps from the pad pass to a shoulder-piece looped round the opposite shoulder and axilla, as in *Fig. 41*.

Rest in bed for three weeks at least after the operation is essential. After that period, exercise can be started with extreme care if the condition of the opposite lung, of the temperature, and of the pulse justify it. Sanatorium régime should be carried on all the time.

**After-results.**—The suddenness with which symptoms change is often dramatic. One of the author's patients lost completely, from the moment of the operation, the cough and sputum which had been an ever-present reminder for over two years. Another lost with equal abruptness the intense stretching drag on the trachea which produced on coughing a sensation of



FIG. 42.—*Case 1*. Age 31. Six months after thoracoplasty.

suffocation. The loss of cough and expectoration is not, however, always so abrupt. The sputum may even be at first increased, but diminish rapidly later. Dyspnoea is occasionally observed during the first week or two. Occasionally pain down the arm, in the shoulder, and in the side of the chest, due to drag on the brachial plexus, is a troublesome feature for a few days. Sometimes the pain is along the course of one intercostal nerve which has escaped the alcohol and is being pressed on. As a general rule, however, it will be found that the post-operative discomforts are, like the shock, surprisingly little considering the character and severity of the operation.

The signs may disappear with equal rapidity, but in some cases the lung, particularly the upper part of it, seems filled with coarse or fine moist sounds which may persist for months. These may be present without there being

any cough or sputum. As Alexander says, "*The compressed lung, even after its disease has become wholly arrested, presents the physical signs of active tuberculosis*". And again later, "*For years the compressed lung, even in the total absence of catarrh and sputum, may be full of coarse and fine râles and a variety of bizarre noises*".

If movements of the arm are begun soon enough, there should be practically no limitation of any normal movement (*Fig. 42*). There is remarkably little deformity of the body; none at all need be visible when the patient is dressed. The shoulder girdle is unaffected, but occasionally there is some lowering, and at times even raising, of the shoulder. Owing to the separation of muscle attachments to the posterior parts of the ribs and of some of the ligamentous bands, there is at first weakness of the muscles on that side of the spine, and

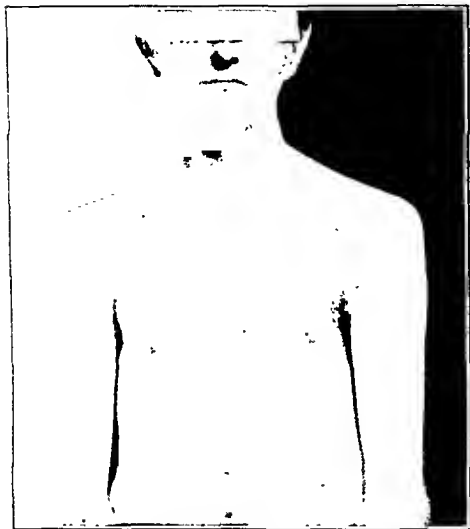


FIG. 43.—Same case as *Fig. 42*.

consequently a tendency to scoliosis concave to the sound side. In chronic cases, however, there has been, prior to the operation, some scoliosis concave to the affected side. The one, therefore, tends to neutralize the other.

Viewed from in front, the patient will exhibit considerable flattening of the affected side, while the junction of the axillary fold and the arm, instead of being pointed, is rectangular (*Fig. 43*). Viewed from behind, it will be seen that the scapula has sunk so that its dorsal surface is on a level with the transverse processes. There is also a hollow round the lower angle and the axillary border of the scapula (*Fig. 44*).

*Fig. 45* shows the X-ray appearances of a case before operation, and *Figs. 46-48* are skiagrams taken after operation.



FIG. 44.—Same case as *Fig. 42* viewed from behind.



FIG. 45.—Case 1. Skiagram taken before operation.



FIG. 46.—Case 1. Skiagram taken six weeks after thoracoplasty.

**Results of Individual Surgeons.—**

Sauerbruch, out of 381 cases, had a mortality during the first week of 14 per cent; 35 per cent were practically healed. Brunner, out of 117 cases, had an 11 per cent mortality; 41.9 per cent were practically healed. Gravesen, out of 96 cases, had an 8.3 per cent mortality; 44.8 per cent were much improved or practically healed.

The author has done 25 thoracoplastic operations for bronchiectasis and tuberculosis: 20 of these were for the latter disease. Of this 20, 8 were done between 1913 and 1915 by the author's modification of the Wilms type of operation. One died of pneumonia 6 weeks after the first stage; one a year

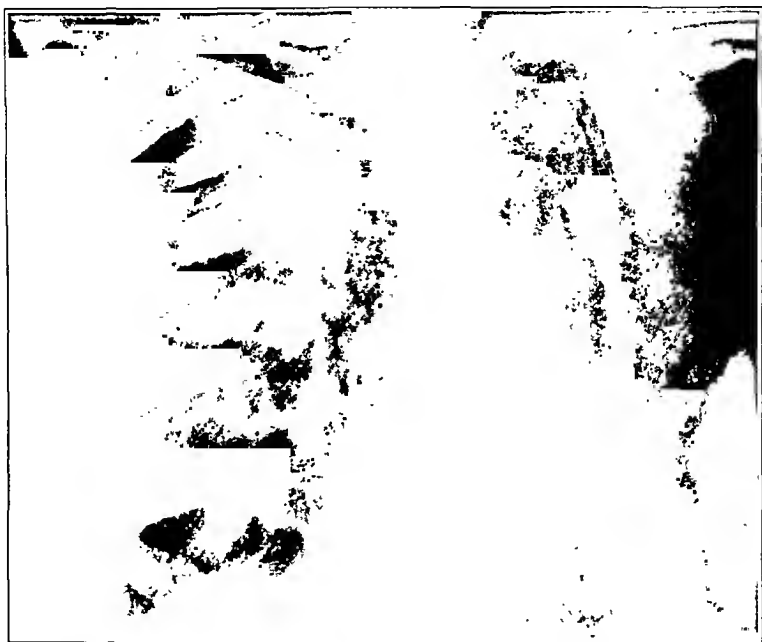


FIG. 47.—Case 2. Age 46. Skiagram taken eight weeks after thoracoplasty (anterior view).

after the operation; one, who from a bedridden invalid had been restored so that she could lead an active life, died 3 years later from extension in the originally sound side, following influenza. Three (one much improved and two clinical cures) were lost sight of 10 months,  $1\frac{1}{2}$  years, and  $3\frac{1}{2}$  years later. Two were alive and well 9 and 11 years respectively after the operation.

The later series includes the 12 cases operated on from four years to six months ago. Of these, one died immediately after the operation (this, the only immediate post-operative death, was complicated by a very rapidly recurring pyopneumothorax); one died 11 months after from tuberculous enteritis; one case, after doing excellently well for  $3\frac{1}{2}$  years, had influenza and developed a basal effusion on the 'sound' side which is proving resistant to treatment; one case has had extension of the disease in the opposite lung.

The remaining eight are all very greatly improved, and four of these are free from all symptoms.

Alexander has collected 1025 cases reported during the six years 1918-23. These show: Cured, 32 per cent; improved, 26 per cent; worse and early and late deaths, 35 per cent; miscellaneous, 7 per cent.

#### **Preliminary Phrenic Evulsion.—**

Sauerbruch recommends that the dome of the diaphragm on the affected side should be paralysed by phrenic evulsion prior to the extrapleural thoracoplasty, in order to test the 'sound' lung and its capacity to tolerate the

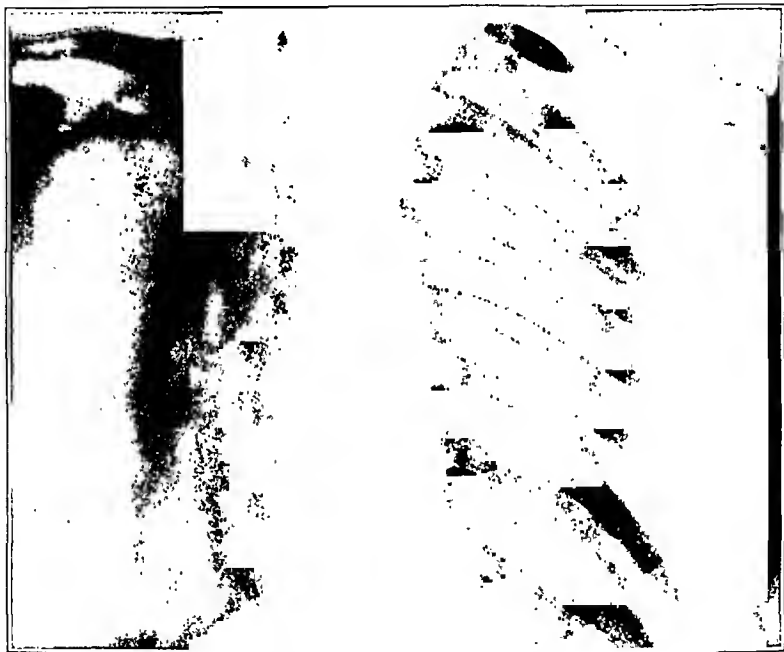


FIG. 48.—Case 2. Same case as Fig. 47 (posterior view).

strain which will be thrown on it by the major operation; also because the paralysis of the dome and its heightened position in the thorax increase the collapse of the base. Hans Alexander is also of the opinion that phrenic evulsion should precede thoracoplasty. Brauer considers this preliminary operation unnecessary and purposeless, as the test can equally well be done by an injection of tuberculin. The author's experience is that the collapse of the base of the lung is usually quite sufficient without the added paralysis of the diaphragm, and that the disturbance produced by this operation is scarcely sufficient to serve as a guide to the responsive capabilities of the 'sound' lung. Phrenic evulsion is undoubtedly of great value in certain cases in helping to improve the patient's general condition before the major operation, especially where there is a constant distressing cough due to irritation of the diaphragm. In all cases of tuberculosis complicated by effusion, the

diaphragm should be paralysed on the affected side so as to reduce the size of the intrapleural cavity.

### Supplementary Operations.—

These are sometimes necessary when stiff-walled cavities in the apex of the lung interfere with the efficient collapse of the chest wall after thoracoplasty. Bull, to remedy this, practises apicolysis, either at the time of the operation, or subsequently.

Brunner does not approve of apicolysis because of the danger of tearing the wall of the cavity, and of the consequent secondary infection. He considers that it is preferable to remove the anterior parts of the ribs overlying the cavity. Claus advocates that these cavities should be laid open.

In conclusion, I will quote the words of Lilienthal, who "considers that the marvellous improvement that takes place soon after the operation (of thoracoplasty) is quite extraordinary. Extrapleural thoracoplasty has the great advantage of fixation of the chest in addition to setting at rest the lung itself".

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**PYELOGRAPHY IN POLYCYSTIC DISEASE OF THE KIDNEYS.**

By W. M. CARRICK, HAMILTON, ONTARIO.

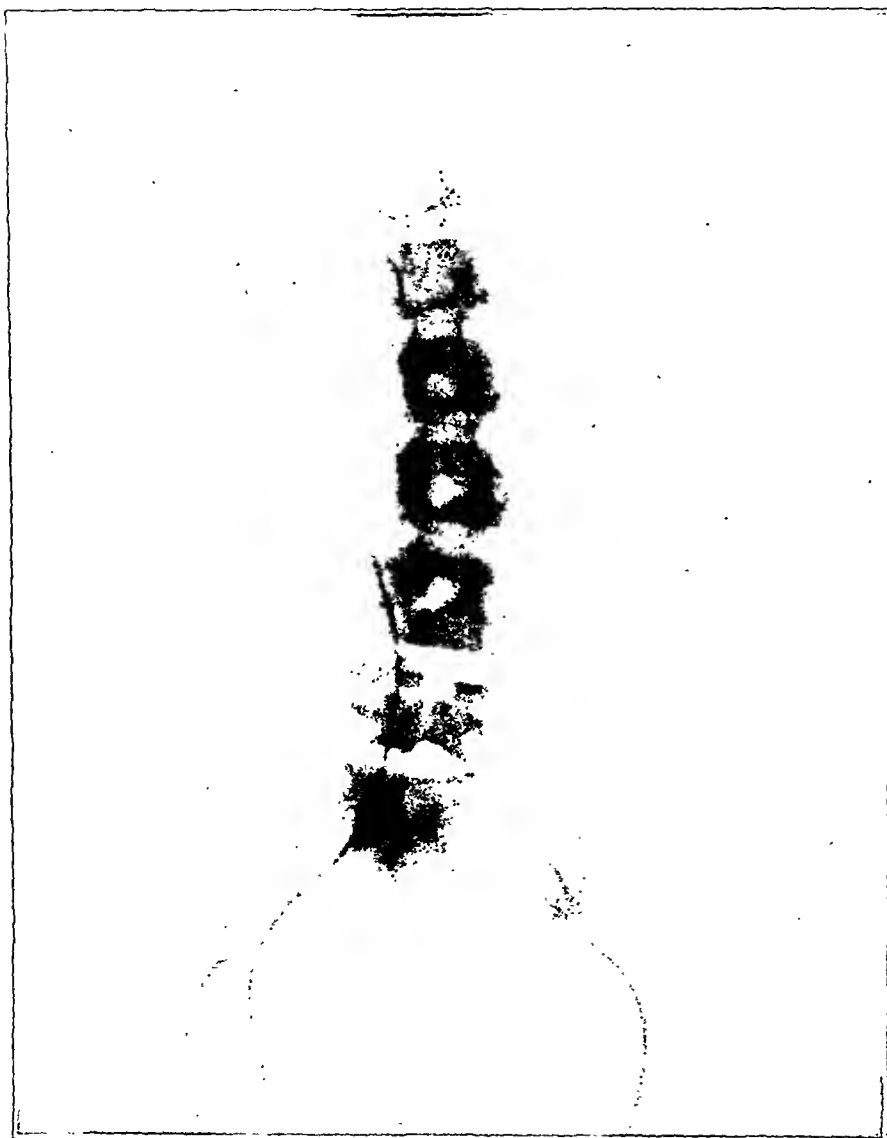


FIG. 49.—Case 1. Skiagram showing injection of left kidney.

IN the October, 1924, issue of THE BRITISH JOURNAL OF SURGERY there appeared a very comprehensive article on polycystic disease of the kidneys,

with reports of five cases. The author had very little to say regarding pyelography in this condition, and I think the accompanying illustrations of two cases might be of interest to show the value of pyelograms in corroborating clinical findings.



FIG. 50.—*Case 2.* Skiagram showing both kidneys injected.

In *Case 1* (*Fig. 49*) the condition existed on both sides, but it was not considered necessary to inject the right kidney. It will be seen that the

right ureteral catheter has doubled on itself and coiled in the kidney pelvis, denoting considerable enlargement.

In *Case 2* (*Fig. 50*), both sides were injected, the left kidney being imperfectly filled owing to rapid return flow of the opaque sodium iodide solution into the bladder.

Both were female patients; one 48 and the other 30 years of age.

Phenolsulphonephthalein efficiency tests were unsatisfactory in both cases, and the blood chemistry done in one case showed: urea nitrogen 49.4, creatin 3.105, sugar 0.070.

## OBSERVATIONS ON THE ETIOLOGY AND TREATMENT OF CYSTITIS.

BY H. W. B. CAIRNS, LONDON.

THERE is a large volume of evidence to show that the bladder is highly resistant to infection by pyogenic bacteria. Rovsing<sup>7</sup> found that injection of virulent bacteria into the normal bladder did not produce cystitis. Panton<sup>6</sup> and others have shown that pathogenic bacteria could frequently be recovered from the urine of individuals, healthy and otherwise, who were quite free from inflammatory lesions of the urinary tract. In addition, there is indirect clinical evidence which points to the same conclusion, namely, the rarity of cystitis after the passage of instruments into the bladder. Thus, at the Genito-urinary Department of the London Hospital, in the last five years it is extremely doubtful whether any case of cystitis has occurred which could be ascribed to infection at cystoscopy. We admit that our technique is good, but we do not flatter ourselves that, during this period in which over 3000 cystoscopies have been performed, we have been able in every case to prevent bacterial infection of the bladder. It is, therefore, clear that in addition to bacterial infection some other factor is necessary for the production of cystitis.

This study was undertaken in order to determine the principles which govern the etiology of cystitis, and in order to find, if possible, some simple classification which would be of use in the investigation and treatment of the disease. The chief results may be summarized as follows: There were two main clinical types of cystitis; one in which the disease tended to clear up quickly, either spontaneously or with very simple treatment; and the other a type in which the disease became chronic. It was found that in almost all cases of the chronic group the cystitis was maintained either by a persistent infection from the kidney or by the presence of residual urine in the bladder.

No claim is made that these observations open up fresh ground; but the writer has found the conceptions developing from this investigation of great service in the management of urinary infections. Like Dr. Johnson's poet, "he feels what he remembers to have felt before, but he feels it with a *great increase of sensibility*". Among modern writers the influence of persistent kidney infections has been stressed by Kidd,<sup>5</sup> while Curtis<sup>2</sup> has shown the importance of residual urine in the production of cystitis in women; and both of these factors have been given their due in cystitis by Caulk.<sup>1</sup> The facts are, however, imperfectly dealt with in most of the current text-books; they are hidden amid lengthy descriptions of 'acid and alkaline cystitis', the morbid anatomical varieties of cystitis ('cystitis cystica', 'granular cystitis', etc.), the bacterial varieties of cystitis, and other questions of secondary importance. Judging, too, from our experience at the London Hospital, the

principles which govern the etiology of cystitis are not properly understood by many practitioners. These deficiencies can only be made good by a consideration of cystitis in terms of disordered function.

### MATERIAL.

An examination was made of the records of the Genito-urinary Department of the London Hospital for the years 1921-24 inclusive. The following cases were excluded :—

1. Cases of tuberculous cystitis.
2. Non-bacterial cystitis due to chemical irritants.
3. 'Purpura of the bladder'.<sup>4</sup>
4. Cases of pyelitis in which, at cystoscopy, only the ureteric orifice and a small adjacent zone of the bladder wall showed signs of inflammatory change. These, although technically cases of pyelocystitis, are for practical purposes uncomplicated cases of pyelitis.

5. Cases labelled in the records 'slight basal cystitis', in which the urine was sterile and free from pus. It was considered that the evidence in these cases was insufficient to justify their inclusion. At cystoscopy the base or trigone of the bladder has normally a bright-red appearance due to its liberal supply of blood-vessels. In cases of prostatic disease, pregnancy, and cystocele, these vessels frequently become engorged, and the resulting appearance may resemble closely the redness of inflammatory hyperæmia. In general, however, it may be said that in the redness due to engorgement a careful examination will show that each individual blood-vessel can be distinguished, whereas when the redness is due to the hyperæmia which connotes cystitis the individual vessels cannot be seen—the surface of the trigone presents a homogeneous red blush.

When the above groups were excluded, 86 cases remained. The diagnosis of 'cystitis' in these cases rested on the evidence obtained from direct examination of the bladder, by cystoscopy in 83 cases, by suprapubic cystotomy in 1 case, and by necropsy in 2 cases. In the severe cases cystoscopy was not performed, save in exceptional circumstances, until the acute symptoms had subsided.

### BACTERIOLOGY.

All cases had pus in the urine. The results of cultivation of the urine (*Table I*) showed an overwhelming preponderance of *B. coli* as the infecting organism, and the results are in accord with those of other writers.

*Table I.*—ORGANISMS CULTIVATED FROM THE URINE.  
(No cultures were made in 14 cases.)

	CASES
<i>B. coli</i> alone ..	40
<i>B. coli</i> and <i>B. proteus</i> ..	2
<i>B. proteus</i> alone ..	3
<i>Staphylococcus aureus</i> ..	1
<i>Staphylococcus albus</i> ..	17
Cultures sterile ..	9
Total ..	72

In 35, or 41 per cent, of the cases, the clinical course indicated that the cystitis was secondary to infection of the kidney or kidney pelvis. In all the other cases it was impossible to determine accurately the source of the infection. Cystitis of the resolving type was associated with prostatitis in a few instances, but the relationship between these foci of infection was not clear.

### CLINICAL TYPES.

The after-histories of 76 out of the 86 cases were traced. A preliminary study of the traced cases showed that they fell into two clearly defined groups :—

1. *Resolving Group*.—Twenty-one cases\* showed a strong tendency towards spontaneous resolution. Many of them were already clearing up before they had had any treatment, and all of them were cured, within four to six weeks of their onset, by extremely simple forms of treatment, such as the administration of potassium citrate and large quantities of fluid by mouth.

2. *Progressive or Protracted Group*.—Fifty-five cases\* showed a tendency to progress. None responded to simple forms of treatment. Many of them had lasted for years. In two cases the cystitis was followed by ascending pyelonephritis and death.

In seeking an explanation for this remarkable division into resolving and progressive types a closer examination of the progressive group showed that in all except one case—a case of solitary ulcer of the bladder—one or other of the following factors was present : either there was a persistent infection of the bladder from the kidney ; or the bladder infected was one which had been, prior to infection, the subject of disordered function. The influence of these factors on the chronicity of the cystitis was amply demonstrated by the success of treatment directed against them.

### THE RETENTION FACTOR IN PROGRESSIVE CYSTITIS.

The type of derangement of bladder function which predisposes to chronicity in cystitis is the inability of the bladder to empty itself completely at each act of micturition ; that is to say, there is always a certain amount of residual urine in the bladder. This, which may be termed the 'retention factor', can be produced by a number of different conditions.

Out of the 55 cases of progressive cystitis there were 30 in which chronicity was due to the retention factor (*Table II*).

The appearance of the bladder and the clinical course in progressive cystitis varied according to the severity of the retention factor and the length of time through which it had been acting. Three main types could be distinguished : (1) *Fulminating type* ; (2) *Severe chronic type* ; (3) *Mild chronic type*.

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\* These figures do not give the true relative frequency of the different types of cystitis, for many cases of the resolving type clear up so quickly on medicinal treatment that they are never sent to the urologist.

Table II.—CAUSES OF THE 'RETENTION FACTOR' IN  
PROGRESSIVE CYSTITIS.

	CASES
Spinal-cord lesion (injury) .. ..	1
Atony of the bladder; old spinal cord lesion ..	1
Vesical calculus .. ..	1
Diverticulum of bladder .. ..	2
Enlarged prostate .. ..	3
Urethral stricture .. ..	2
Urethral stricture, diverticulum and stone of bladder .. ..	1
Urethral stricture and atony of bladder ..	2
Urethral stricture and enlarged prostate ..	1
Urethral stricture and bilharziasis of bladder ..	1
Cystocele .. ..	14
Retention of urine of unknown origin ..	1
Total .. ..	30

1. **Fulminating Type.**—Progressive cystitis was seen in its severest form when the pyogenic infection occurred in a bladder which was the subject of an unrelieved acute retention. There were two such cases in this series, and both of them were fatal.

*Case 1.*—F. E., a male, age 42, was hit on the head by a falling sack. He sustained a fracture-dislocation of the spine, and the spinal cord was completely divided at the level of the third thoracic segment. He developed retention of urine. A catheter was passed once daily on the second, third, and fourth days of the illness, and twice on the fifth day. On the sixth day the urine was blood-stained; a catheter was then tied in, and the bladder was irrigated frequently. The patient died on the eleventh day of the illness.

Post-mortem examination revealed, in addition to the spinal lesions, acute hæmorrhagico-purulent cystitis, ascending pyelonephritis, and septicæmia.

*Case 2.*—D. J., a female, age 18, had suffered from weakness, drowsiness, and incontinence of urine for a fortnight. Three days before admission to hospital she had become delirious and had had 'convulsive fits'. The bladder was palpable 1 in. above the pubes. She was mentally incoherent and had weakness of the right side of the face, and weakness and loss of reflexes of the lower extremities. A diagnosis of encephalomyelitis was made. The patient gradually passed into a state of deep coma and died.

Post-mortem examination showed that death was due to surgical uræmia following ascending pyelonephritis. A small stone was lying in the base of the bladder. This had evidently been impacted at the neck of the bladder, thereby producing retention of urine. For, at the vesical outlet, the mucosa was necrotic over an area corresponding to the size of the stone. The rest of the bladder was in a condition of severe hæmorrhagico-purulent cystitis.

In both these cases, examples of widely different diseases, the first common factor was retention of urine. The retention preceded and was directly responsible for the onset of the cystitis, and the cystitis was followed, in each instance, by a fatal ascending pyelonephritis.

Fulminating cystitis may supervene in any variety of acute retention, but it is usually seen in cases in which, for various reasons, the retention has developed without giving rise to acute discomfort in the bladder region. It is probably on account of this very absence of pain that the urgency of such retention is apt to be disregarded. In some cases—for example *Case 9*—



distention of the bladder may even be overlooked because there is an associated incontinence of urine.

The transition of a case from a condition of simple retention to one of retention plus fulminating cystitis is extremely insidious. It is only when the kidneys have become secondarily infected that the definite signs appear. The earliest of these are most commonly fever, drowsiness, wasting, pallor, diarrhoea, and pain in one or other loin. By the time these signs have become apparent the condition of the patient is well-nigh hopeless; permanent damage has been done to the kidneys, and death is almost inevitable. Cystoscopy should never be performed in these cases, for the bladder wall is very friable, and the passage of the cystoscope is liable to produce perforation and extravasation of urine.

It is thus clear that little success can be expected in the diagnosis and treatment of fulminating cystitis. But the onset of this condition can be easily and surely prevented if every case of distended bladder, no matter how mild the symptoms which accompany it, is regarded as an emergency. Too much stress cannot be laid on the fact that every distended bladder should be emptied as rapidly as is consistent with safety; and, furthermore, urine should not be allowed to re-accumulate until the normal function of the bladder has been restored. If this cannot be done satisfactorily by indwelling catheter, suprapubic cystotomy should be performed without delay.

**2. Severe Chronic Type.**—The second type of retention cystitis comprised a group of cases in which cystoscopy revealed signs of severe inflammation of the whole of the bladder mucosa. The normal difference between the trigone and the rest of the bladder had disappeared. The surface of the bladder had, for the most part, a homogeneous velvety-red appearance; the outlines of the trabeculae and the interureteric bar were usually obscured, and the ureteric orifices were represented by mere chinks, not easily found. Submucous hemorrhages were sometimes seen, particularly around the neck of the bladder. In addition, it was not uncommon to find large sharply defined patches of white membrane, resembling a thin film of sugar icing, spread over the surface of the inflamed mucosa; such areas usually occurred on the base of the bladder. Attached to the surface of the bladder there were often numerous flakes of fibrino-purulent material, which in the more advanced cases appeared, particularly on the fundus, in the form of large white shaggy masses projecting into the lumen of the bladder. These masses were such a striking feature and were so invariably restricted to cases of long-standing retention that we have come to use a special term 'shaggy cystitis' for this type of progressive cystitis. To some extent, 'shaggy cystitis', may resemble extensive ulcerating and necrosing carcinoma of the bladder, a resemblance which was, in the cases of this series, supported by the history of attacks of hæmaturia. In the cystitis cases, however, the masses were more densely white than in carcinoma, and they were entirely avascular. In addition they were much more freely movable; they waved about in the fluid medium; and after repeated bladder irrigations they became detached and nothing beyond an inflammatory change was found in the subjacent bladder wall. The hæmaturia coincided with the detachment of the shaggy masses.

Such appearances indicate a considerable inflammatory hyperæmia and swelling of the mucous membrane, sometimes associated with a fibrinous or fibrino-purulent exudate, which may remain attached to the bladder wall in the form of a membrane or a shaggy mass.

The syndrome produced by this type of cystitis is as follows. The patient, usually a male, states that for years he has had urinary trouble, and that in the last year or so he has been worried by great nocturnal and diurnal frequency. He has frequently been content to put up with this state of affairs until an attack of hæmaturia occurs which impels him to seek treatment. The urine is turbid and contains large flakes of débris; it is alkaline, and smells—so, indeed, does the patient—of ammonia. Residual urine is invariably present; it usually exceeds 5 oz., and the bladder may be palpable in the hypogastrium. It is necessary to spend up to half an hour irrigating the bladder before the medium becomes clear enough for cystoscopy; in fact, a good view of the bladder wall may not be obtained until the bladder has been irrigated on several successive days. The appearances that will then be seen have already been described.

There were eleven examples of severe chronic cystitis in this series. Different cases were preceded by a wide variety of urinary disorders, which had, however, one thing in common, namely, the presence of residual urine in the bladder. It was this retained urine, acting through a long period of time, which had been responsible for the production of the cystitis.

The commonest primary causes of residual urine were urethral stricture, diverticulum of the bladder, disease of the spinal cord, and enlargement of the prostate. The actual etiology of each particular case was, however, usually complicated, as can best be shown by the cases in which the primary affection was a urethral stricture.

*Case 3.*—Urethral stricture: diverticula of bladder: vesical calculi: 'shaggy cystitis'.

F. S., a male, age 56, had had gonorrhœa in 1894. In 1919 he had developed acute retention, due to a calculus impacted behind a stricture of the membranous urethra. Dilatation of the urethra and lithotomy were performed. In 1923 the stricture had become troublesome again, and an internal urethrotomy was done. He was admitted under the care of Mr. Hugh Lett in 1924 complaining of frequent and painful micturition and attacks of hæmaturia.

The urine contained a large amount of pus, and on culture yielded *B. proteus*. X-ray examination showed three vesical calculi, two of which were later found to occupy diverticula; there were also numerous small prostatic calculi. At cystoscopy, in addition to 'shaggy cystitis', there were numerous shallow pouches in the bladder wall and one which was evidently deep. The residual urine measured 2 oz.

At operation, by Mr. Lett, the stones were removed by suprapubic cystotomy and the bladder was drained. Daily irrigation of the bladder was carried out for three weeks, and then the suprapubic wound was allowed to heal. On discharge from hospital his micturition was normal and there was no residual urine; he "felt better than he had done for months".

*Case 4.*—Urethral stricture: atony of bladder: 'shaggy cystitis'.

J. S., a male, age 67, had had gonorrhœa in 1884 and external urethrotomy for urethral stricture six years later. In 1924 he presented himself at the Genito-urinary Department complaining that for one year he had had great frequency of micturition and attacks of hæmaturia.

There was a urethral stricture which required dilatation. When this had been

done, cystoscopy revealed a typical 'shaggy cystitis'. The residual urine on several occasions varied between 30 and 40 oz. There was no sign of prostatic enlargement or of nervous disease, and the large amount of residual urine, even after dilatation of the urethral stricture, could only be attributed to atony resulting from chronic over-distention of the bladder. This view was confirmed by the appearances observed at cystoscopy; when the bladder contained only 10 oz. of fluid its lateral wall and fundus were seen to sag in towards the centre of the viscus—in other words, there was a complete loss of 'shortening reaction' (Sherrington<sup>8</sup>).

The patient was taught to catheterize himself, and the bladder was irrigated once a week. He was greatly improved.

**Case 5.—Urethral stricture: old bilharziasis of bladder: progressive cystitis.**

W. R., a male, age 43, had contracted bilharziasis of the bladder in South Africa in 1901. Two years later he had gonorrhœa. In 1909, after a course of treatment, he was told that he was cured of bilharziasis. In 1913 he began to suffer from occasional hæmaturia and frequency, which increased to such an extent that he had to wear a portable urinal. In 1919 he underwent a course of dilatation for urethral stricture; this relieved but did not cure him, and the frequency was still considerable. Hæmaturia returned in 1921, and he was admitted to hospital under the care of Mr. Lett.

No ova were found, but the urine yielded *B. coli* on cultivation. Cystoscopy showed great swelling and hyperæmia of the whole of the bladder mucosa. The bladder capacity was 'small', and the residual urine measured 3 oz.

The bladder was drained through a suprapubic opening, and washed out daily. After one month the opening was allowed to close, and the patient was discharged from hospital considerably improved.

There were two other cases of severe progressive cystitis and urethral stricture. As in the cases described above, residual urine was present, and was due to a combination of conditions. In one the stricture was associated with atony of the bladder, and the residual urine measured 10 oz. The other, a patient who had 15 oz. of residual urine in the bladder, had had stricture trouble for years, and now had in addition considerable prostatic enlargement. These two cases received great benefit from regular dilatation, catheterization, and bladder irrigations.

Chronic progressive cystitis was also seen in two cases of enlargement of the prostate. In one the residual urine measured 20 oz. Prostatectomy was performed and the cystitis disappeared. The patient was seen again two years after the operation, when his urine was clear and the frequency was normal; in addition he was able to empty his bladder completely and there was no residual urine.

The second case of enlarged prostate demonstrated how, in retention cystitis, pus and bacteria tend to concentrate in the residual portion of the bladder contents, thereby aggravating the cystitis.

**Case 6.—Enlarged prostate: progressive cystitis.**

L. B., age 49, had suffered for eighteen months from frequency and dysuria. One year before he came to the Genito-urinary Department he had been partially relieved by lithotripsy, but the frequency had still continued. He had had gonorrhœa fourteen years before.

He passed 8 oz. of slightly turbid urine. A cystoscope (22 Charrière) was then inserted and 8 oz. of residual urine were withdrawn. This urine was quite different in character from the first specimen; it was thick and yellow, and when it had stood for a few minutes a deposit of one-half volume of pus formed. The bladder mucosa showed a severe generalized cystitis and numerous small shaggy

masses on the mucosa of the neck. The lateral lobes of the prostate were seen to be greatly enlarged.

A similar observation may sometimes be made at the operation of suprapubic cystotomy for retention of urine and enlarged prostate when cystitis is also present. The urine which escapes when the bladder is opened is almost or quite clear, whereas that which comes later from the bottom of the bladder contains a large amount of pus. It is therefore evident that stagnation of pus and bacteria occurs in bladders which cannot empty themselves completely. Even when, as in *Case 6*, the bladder is capable of expelling half its contents, the forcible contractions of the bladder detrusor which are necessary to such an act do not bring about admixture of the contents. The same urine may remain in the bladder for days at a time although the patient is passing water with comparative freedom. This residual urine becomes loaded with pus, and is in effect a focus from which further infection of the bladder is continually occurring. Bladder irrigations are of the greatest benefit in retention cystitis, and the manner in which they act is readily understood in the light of the observations made in *Case 6*. By washing out the bladder until the fluid is returned clear the infective residual urine is removed.

There were two cases of diverticulum of the bladder in which advanced cystitis was present. The diverticula in both cases opened in the base of the bladder immediately behind the ureteric orifice. The situation of the diverticula appeared to be a point of some importance, for, during the period under consideration, several other cases of bladder diverticulum were observed in which there was practically no cystitis at all, and in these cases the diverticulum was always situated at the fundus of the bladder. That is to say, when a diverticulum opens in a dependent part of the bladder, stagnation of urine occurs within it and cystitis is accordingly produced; whereas in the case of a fundal diverticulum there is no stagnation of urine and, consequently, no cystitis. If man stood on his head instead of on his feet the basal diverticula would then be the innocuous ones. Possibly postural treatment may be of value in cases of basal diverticula in which operation for some reason cannot be carried out; but I have had no experience of this form of treatment, nor have I found any reference to it in the literature.

One of the diverticulum cases illustrated another retention factor which may act in progressive cystitis: severe cystitis itself, it appears, can aggravate a pre-existing retention.

*Case 7.*—Persistent suprapubic fistula after suprapubic lithotomy: diverticulum of bladder: chronic progressive cystitis.

F. S., age 27, was admitted under the care of Mr. Lett, on account of a persistent suprapubic fistula which had followed the removal of a vesical calculus elsewhere six months before. One attempt had already been made to close the fistula by operation, without success.

He was treated by indwelling catheter and daily bladder irrigations for six weeks, at the end of which period the suprapubic wound had closed. Cystoscopy then showed severe general cystitis and, behind the mouth of the right ureter, the orifice of a diverticulum. The bladder irrigations were continued, but the catheter was now removed and the patient was encouraged to make water. He tried very hard, but seemed quite unable to do so, and the suprapubic fistula re-opened. After another period of treatment by indwelling catheter the fistula healed, but when he

tried to pass water naturally he was again unable to do so and the urine came through the wound. Accordingly another operation was performed, at which the diverticulum was removed; this involved resection of the lower part of the right ureter, the upper cut end being transplanted into the adjacent bladder wall. The patient died six days later of ascending pyelonephritis. At necropsy the spinal cord was found to be normal.

In this case there was no mechanical obstruction to the outflow of urine, and the nervous paths were normal. The acute retention must therefore have been due to some disturbance of the bladder musculature, probably spasm of the sphincter, which was brought on by the pre-existing cystitis. *Case 8* in its later stages also showed that cystitis by itself may produce retention. Similar observations have been made by Head and Riddoch<sup>3</sup> in cases of spinal injury: They state that "long-continued cystitis tends . . . at any rate in the early stages, to decrease the completeness with which it [the bladder] is evacuated". Thus it appears that a vicious circle may be established; residual urine is responsible for the production of cystitis, which may in turn produce further retention of urine, and this aggravates the cystitis. The only way in which such a circle can be broken is by complete drainage of the bladder.

There was one example of progressive cystitis associated with spinal-cord disease. This case was of great interest because it remained something of a mystery until our study had advanced so far as to reveal to us the true value of the retention factor in the production of cystitis. The case as it presented itself to us was as follows:—

*Case 8.*—Old tuberculous disease of the spine: atony of bladder: progressive cystitis.

T. G., age 64, complained of frequent, difficult, and painful micturition. He had had this trouble for an indefinite number of years, but it had been getting much worse in the last two months. As a young man he had had gonorrhœa, and ten years before he came to the Genito-urinary Department he had had spinal caries; as a result he had lost  $4\frac{1}{2}$  in. in height, and had now an extensive kyphosis in the dorso-lumbar region.

The cystoscope (22 Charrière) was passed without any difficulty. There was general hyperæmia and swelling of the bladder mucosa and a very slight intravesical enlargement of the lateral lobes of the prostate. The residual urine measured 3 oz. and the bladder capacity only 5 oz.

Here was a case of cystitis which was manifestly not associated with enlarged prostate or urethral stricture. When it was realized that the residual urine was the key to the situation a further search was made for some pre-existing cause of retention. None of the usual signs of nervous disease was present, but a closer inquiry revealed that he had had paraplegia when he was ill with spinal caries, and that the symptoms of cystitis had come on after that. The case was now clearly understood. At the time of the spinal caries some permanent damage had been done to the nervous paths for micturition in the lumbo-sacral portion of the spinal cord. The reflexes governing the act of micturition were thenceforward impaired, and chronic retention of urine ensued and was responsible for the onset of cystitis. The case also showed that cystitis acting through a long period of time may cause great reduction of the bladder capacity.

There was one case of severe progressive cystitis in which the retention factor was of somewhat obscure origin.

*Case 9.*—Retention of urine (? cause): progressive cystitis.

E. P., age 20, came to the London Hospital on account of incontinence of urine. The bladder was distended, the fundus being palpable 1 in. below the umbilicus, yet he was wearing, and had for the past fortnight worn, a portable urinal! He was admitted at once.

Three months before, he had had an obscure febrile illness which was at first thought to be typhoid fever; this diagnosis proved to be incorrect, for his serum never agglutinated either *B. typhosus* or *paratyphosus A* or *B*, and the febrile symptoms cleared up within ten days. Towards the end of this illness retention of urine developed. The retention was relieved once or twice daily by catheter, and at the end of a week he passed water again, small amounts at frequent intervals. At this time the urine contained a little blood and a lot of 'matter'. In the absence of any other demonstrable cause of retention, the prepuce, which was phimotic, was removed. The operation did not cure his trouble; frequency and hæmaturia continued and, later, incontinence of urine developed. In addition he began to have unquenchable thirst and a constant aching pain in the hypogastrium. He then came to the London Hospital, and was admitted under the care of Mr. Lett.

The urine was alkaline, contained blood and pus, and yielded *B. proteus* on cultivation. Examination of the urinary tract for calculi was negative. No abnormality of the nervous system was detected.

A catheter was tied in; the bladder was slowly emptied, and thereafter irrigated twice daily with a 1-4000 solution of oxycyanide of mercury. Some trouble from blockage of the catheter occurred at the beginning of this treatment, and his condition became very critical. He began to have pain in the right loin, uncontrollable diarrhœa, and occasional vomiting. The temperature rose to 102°, the tongue was dry and coated, and his features took on a sunken, pinched appearance. In addition he became drowsy, languid, and increasingly pale. It was clear that he was developing ascending pyelonephritis.

Prompt attention to the catheter and increase in the number of daily bladder irrigations averted a catastrophe, and in a short time the patient began to improve. Cystoscopy a fortnight after he was admitted to hospital showed extremely severe hyperæmia and swelling of the bladder mucosa, numerous large white shaggy masses, and a few submucous hæmorrhages.

He was treated for a further three weeks by indwelling catheter and bladder irrigations, at the end of which time the bladder inflammation was seen by cystoscopy to be recovering. The catheter was then removed, but was passed once daily for bladder irrigation until there was no longer any residual urine. When discharged from hospital two months after admission his urinary functions were normal and he was free from symptoms.

There was no abnormality of the prostate, urethra, or nervous system in this case, and it was evident that the retention must have arisen as a result of the febrile illness or the phimosis. The retention preceded and caused the cystitis, and a vicious circle was then established; for, after the original cause or causes of retention had been removed, the bladder remained in a condition of acute retention, a condition which can only have been due to the cystitis. If complete drainage of the bladder had not been promptly established and maintained, the patient would have died of ascending pyelonephritis.

It will be convenient to review briefly the cases of severe progressive cystitis which have been described above.

A number of different pathological conditions gave rise to cystitis. In

each case the cystitis was preceded by a partial retention of urine which was an expression of the derangement of bladder function produced by pre-existing disease.

When the cause of the retention could be removed, the recovery from cystitis was complete.

When, in the cases in which the function of the bladder had been irreparably damaged, complete drainage of the bladder was established, the cystitis tended to clear up at once; the completeness of the cure in these cases was only limited by our inability to remove the cause of the retention and our disinclination to establish permanent suprapubic drainage.

Catheterization and irrigation of the bladder were of great value in these cases; by such measures the bladder was temporarily emptied and washed free of all retained urine.

There is, therefore, a volume of evidence to show that retention of urine is an extremely important cause of chronicity in cystitis. It is clear that cystitis must not be treated empirically. When residual urine is present in cases of cystitis its cause must be sought and removed if the cystitis is to be cured. If the retention of urine is neglected or overlooked, the cystitis may increase in severity and a fatal ascending infection of the kidneys may develop.

**3. Mild Chronic Type.**—In this group have been included cases of persistent cystitis in which the inflammatory changes were almost or entirely confined to the base of the bladder. The usual appearance at cystoscopy was a redness and swelling of the mucosa of the trigone. On this inflamed surface confluent groups of small cysts (cystitis cystica) or papules (granular cystitis), and sharply defined patches of 'sugar icing' were frequently seen. Submucous hæmorrhages occurred occasionally. The symptoms associated with these lesions were frequency, dysuria, and occasionally hæmaturia—of a much milder degree than occurred in severe progressive cystitis. The general health was not seriously lowered, and it was possible to treat practically all of the cases in the Cystoscopy Department.

There were 16 cases of mild progressive cystitis. In all of them there was present some lesion of the urinary tract which prevented the bladder from emptying itself completely at each act of micturition. Urethral stricture was found in one case, enlarged prostate in another, and in the remaining 14 cases a cystocele, usually of minor degree. Residual urine was present in amounts varying from 1 to 3 oz.

When the retention factor could be completely removed, as in the case of enlarged prostate and stricture, the cystitis disappeared. In the cases of cystocele, treatment by a suitable ring pessary was followed by subsidence of the cystitis.

Two practical difficulties arose, however, in the treatment of the cystocele cases. Apparently treatment of the small cystocele by ring pessary is not invariably satisfactory. The ring pessary, it seems, will not control those cystoceles which are unaccompanied by prolapse of the uterus. It is difficult to keep clean, and, if not changed regularly, may produce vaginal hæmorrhage. Thus, in the case of a woman, 67 years of age, a mild progressive cystitis was immediately relieved by the insertion of a ring pessary. When she had worn

it for six months she began to bleed from the vagina, and the pessary was accordingly removed. Two months later she had another attack of cystitis. Thus our first difficulty was that in some cases the logical treatment—namely, the elimination of the retention factor—could not be carried out.

The second difficulty in treatment arose from the fact that often the symptoms were not confined to the urinary system. Some patients exhibited nervous and abdominal symptoms, such as headache, nervousness, mental breakdown, epigastric or general abdominal pain, 'falling of the abdomen' on standing up, and, almost always, a feeling of general weakness. These patients were, in fact, examples of that condition which is at present termed 'visceroptosis'. They differed from the usual type of 'visceroptosis' in that, having developed mild cystitis, their principal symptoms were pelvic rather than epigastric. Pessary treatment was often contra-indicated in these cases.

It will thus be seen that the cure of cystitis with cystocele may be a difficult matter. In this series great improvement sometimes followed the injection of collargol (10 to 20 per cent) into the bladder. Where 'visceroptosis' was present the provision of an abdominal belt seemed to help in alleviating not only the abdominal but also the urinary symptoms. A potassium bromide mixture was also useful in these cases. When the ring pessary could be worn, cure followed. In the other cases complete cure was difficult to obtain and relapses were common.

Before considering the second great factor in the production of progressive cystitis, it is necessary to point out that by no means all cases in which the bladder contains residual urine develop cystitis. Similarly, cases of cystitis, in which there is residual urine, albeit a small amount, may recover without any treatment having been directed against the retention factor. There is, however, no doubt that in all cases of cystitis cure will be most quickly obtained by direct treatment of the retention factor; and, except for the cases which are now to be considered, only those will become chronic in which the bladder contains residual urine.

### PROGRESSIVE CYSTITIS DUE TO PERSISTENT KIDNEY INFECTION.

Of the 55 traced cases of progressive cystitis, 24 were due to infection of the kidney pelvis. The relationship between the kidney lesion and the cystitis was most distinctly shown in cases of pyonephrosis. There were 5 such cases, of which the following is an example.

#### *Case 10.*—Pyonephrosis: severe cystitis: nephrectomy.

M. T., female, age 23, under the care of Mr. H. S. Souttar, had suffered for three weeks from severe pain in the left loin, vomiting, and frequency of micturition.

She looked very ill and her temperature was 102.4°. There was great tenderness and rigidity over the left kidney region. The urine contained a large amount of pus (culture, *B. coli*). She was treated with large quantities of fluids and large doses of alkalis, but she grew steadily worse. Cystoscopy showed a severe cystitis; the whole of the bladder mucosa was intensely hyperemic and oedematous. A continuous stream of thick pus was seen to pour from the left ureteric orifice.

The left kidney was explored. A huge pyonephrosis was found, and the kidney was accordingly removed. The post-operative course was uneventful. Three weeks after the operation the urine was normal except for a trace of albumin and pus. At cystoscopy slight cystitis of the trigone was found, but the remainder of the bladder mucosa had become normal.



The patient was seen again one year after the operation. She had had no urinary symptoms since discharge from hospital and her urine was normal. It must be presumed, therefore, that the recovery was as complete as it was rapid.

This case shows that severe cystitis may be produced by infection from the kidney. It shows, too, how quickly a bladder will recover when once the infective focus is removed.

In the 19 cases of progressive cystitis associated with pyelitis the relationship between kidney and bladder lesions could not be so distinctly proved, for the obvious reason that the kidney focus could not be controlled, as it was by nephrectomy in cases of pyonephrosis. In many cases, however, there was clinical evidence of a pre-existing (and primary) kidney pelvis infection. The illness had been ushered in with pain in one or other loin, associated with high temperature and shivering. In a few cases there were no symptoms of primary pyelitis. In one of these, examination by X rays, which is performed as a routine, showed a stone in the right kidney, and led to the detection of the primary cause of infection.

Evidence obtained by cystoscopy was of very great value. The presence and persistence of signs of inflammation around one ureteric orifice was an indication of infection of the corresponding kidney pelvis. In the following remarkable case this cystoscopic sign gave the only clue to the origin of the cystitis, and it did not appear until the case had been under observation for three months.

*Case 11.—Progressive cystitis: 'concealed' pyelitis.*

A. S., female, age 22, attending the Genito-urinary Department, had had for seven months attacks of painful, urgent, and frequent micturition, associated with hæmaturia. The attacks lasted a fortnight, and came on about once every six weeks. The pain occurred during micturition, in the hypogastrium and at the urethral orifice. There had been no pain in the loins. The urine contained pus and blood. Cystoscopy showed moderate general cystitis, and an area of ulceration between the ureteric orifices.

The patient was treated for three months with injections of collargol (20 per cent) into the bladder. The pain and frequency decreased but did not disappear, and the urine still contained pus and occasionally blood. At cystoscopy it was now seen that there was a patch of ulceration and injection in the neighbourhood of the right ureteric orifice, whereas the rest of the bladder showed little abnormality. Collargol was accordingly injected through a ureteric catheter into the right kidney pelvis. Within a fortnight the symptoms had entirely disappeared and the urine had become clear.

Injection of collargol into the pelvis of the kidney was followed, in this case, by immediate disappearance of the cystitis. The same treatment was similarly effective in other cases of progressive pyelocystitis in which the primary pyelitis was not complicated by pregnancy, stone, or hydronephrosis. In a few cases in which there was definite clinical evidence that pyelitis had preceded cystitis, injection of collargol merely into the bladder was followed by cure. This may have been due to regurgitation of the collargol up the ureters, for Volker<sup>9</sup> has shown that in some cases collargol injected into the bladder can be detected in the pelvis of the kidney a short time afterwards.

From consideration of the evidence given above it is clear that chronicity may be due to a persistent infection from the kidney.

## SOLITARY ULCER OF THE BLADDER.

In an early part of this paper it was stated that of the 55 cases of progressive cystitis there was one the chronicity of which could not be explained by the presence of either a retention factor or a persistent infection from the kidney. This was a case of solitary ulcer of the bladder. The case is a good example of an uncommon type of cystitis the pathogenesis of which is still uncertain.

## Case 12.—Progressive cystitis: solitary ulcer of bladder.

L. S., a Polish Jew, age 35, came to the London Hospital in 1915 complaining that for one year he had had painful and frequent micturition with occasional hæmaturia. At the onset of the illness he had had a urethral discharge for eight days, but this had not recurred. The urine contained pus. At cystoscopy the left lobe of the prostate was seen to be enlarged. The left seminal vesicle also was found by rectal examination to be enlarged. X-ray examination of the urinary tract was negative. A diagnosis of prostatitis and vesiculitis was made. The patient failed to attend for treatment.

He reported again in 1917 with the same symptoms. He was treated for three months by irrigations of the posterior urethra. His symptoms were eased, so he went away before completing his course of treatment.

Since Jan., 1922, he has been attending the Genito-urinary Department occasionally, and at the time of writing (Jan., 1925) he is still under treatment. He suffers from frequency (twice during the night and half-hourly by day), and he has pain in the hypogastrium at the end of micturition. About once every six months he has had an attack of hæmaturia, which is accompanied by increase of frequency and pain. He has never had pain in the kidney regions. General examination reveals nothing abnormal. The Wassermann reaction is negative.

The urine contains small threads, pus, and red blood-corpuscles; cultures are sterile, and examination for tubercle bacilli has been negative on several occasions. There is no residual urine. The bladder capacity is limited to 5 oz.; over-distention of the bladder causes acute discomfort and hæmaturia. The blood comes from an ulcer, about 1 cm. diameter, some distance above the right ureteric orifice. The ulcer has a red base and sharply-cut edges; the surrounding mucosa is perfectly healthy. The prostate is slightly enlarged, and the adjacent bladder mucosa shows slight œdema and injection. The rest of the bladder mucosa and the orifices of the ureters are normal.

This ulcer was observed by cystoscopy three years ago and has been examined many times since then. It has not increased in size, but on the other hand it has never healed in spite of various forms of treatment. The symptoms are considerably relieved by injection of collargol (20 per cent) into the bladder, and the patient is always anxious to have this form of treatment.

## SUMMARY.

1. From the foregoing account it is evident that cases of cystitis fall into two main groups: (a) Those which clear up quickly, either spontaneously or after simple forms of treatment (*resolving cystitis*); and (b) Those which become chronic (*progressive cystitis*).

2. The paths of bacterial infection in resolving cystitis are not completely understood. In some cases the infection undoubtedly comes from the kidney, in others possibly from the prostate. There remain, however, many cases in which it is impossible to determine the source of the infection.

3. Chronicity in cystitis is due to a number of conditions. In rare cases it is due to a solitary ulcer of the bladder, a condition of unknown etiology.

In most cases it is due either to persistent infection from the kidney or to the presence of retained (or residual) urine in the bladder.

4. A persistent focus of infection in the renal pelvis may give no clinical signs. Evidence obtained by cystoscopy is frequently of great value in the detection of such foci. If the lesion of the renal pelvis is treated efficiently, the cystitis will clear up at once.

5. Any variety of retention of urine may produce progressive cystitis. The successful treatment of retention cystitis depends entirely on the establishment of complete drainage of the bladder either by natural or artificial means. Bladder irrigations are of great service in this variety of cystitis.

6. When cystitis occurs in a case of unrelieved acute retention there is grave danger that it will be followed by ascending pyelonephritis, a condition which is usually fatal. When it is realized that all cases of retention of urine require immediate and complete relief, then only will ascending pyelonephritis, the occurrence of which is a grave reflection on the teachings and practice of surgeons of to-day, cease to be common. Drainage of the distended bladder is an operation of greater emergency than that of drainage of a pelvic abscess.

7. Finally, cystitis, though a distinct clinical entity, is usually secondary to some other lesion of the urinary tract. It should not be treated empirically; the exciting and predisposing causes should be sought by a thorough investigation of the whole of the urinary tract. When these causes have been dealt with, the inflammation of the bladder will subside of its own accord.

I desire to thank the surgeons of the London Hospital for permission to study their cases, and further to express my indebtedness to Mr. Hugh Lett, Director of the Genito-urinary Department, and Mr. G. E. Neligan, for their inspiration and advice during the preparation of this paper.

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**RIEDEL'S CHRONIC THYROIDITIS:  
WITH A REPORT OF SIX CASES AND A CONTRIBUTION  
TO THE PATHOLOGY.**

BY A. F. BERNARD SHAW AND R. P. SMITH, NEWCASTLE-UPON-TYNE.

A PECULIAR affection of the thyroid which is of special interest to the surgeon and the pathologist was first described in 1896 by Riedel as a "chronic inflammation of the gland leading to the formation of an iron-hard tumour".

The disease occurs both in men and women, and is characterized by the appearance in the thyroid of an extremely hard tumour. Frequently the whole gland is enlarged, but one lobe only may be attacked. Sometimes a localized nodule is present without general enlargement. The date of onset and mode of development may be difficult to elicit; the swelling may begin on one side and then extend to the rest of the gland, but in some cases it appears to begin diffusely. It may first be noticed by the relatives. The rate of growth is often slow at first, but later becomes accelerated. In a few cases there is a history of fluctuations in size. The swelling seldom forms a bulky tumour, and its comparatively small size compared with the marked symptoms has often been emphasized. Pain is rare. The early cases are without symptoms, but there is a remarkable tendency for the tumour to become adherent to and even to infiltrate the neighbouring structures; it becomes firmly fixed to the trachea, carotid vessels, recurrent nerves, and other structures. The skin, however, is rarely involved. Pressure symptoms cause the patient to seek relief. The symptoms are increasing dyspnoea with suffocative attacks, particularly at night, dysphonia or aphonia, and sometimes dysphagia. The general health is fairly good, and the medical history without special moment. There is no clinical evidence of hypo- or hyperthyroidism.

The most striking feature of the tumour is its remarkable hardness and density, which has been likened by various observers to iron, stone, wood, cartilage, or bone. Usually it is smooth or only finely nodular on the surface. With extensive adhesions the cervical structures are obscured, the margins of the tumour ill-defined, and there is loss of mobility. The lymph-nodes are not enlarged. There is no pyrexia, and a general clinical examination reveals no special abnormality. The clinical picture bears a close resemblance to certain forms of cancer, and most of the cases hitherto described have been diagnosed at first as malignant disease. The cut section of the gland is hard, smooth, creamy-white, opaque, and often intersected by fibrous strands. Microscopically there is no evidence of malignancy.

The only successful treatment, at present, is surgical. Drugs and X rays have proved of no value. Cure has been effected by complete thyroidectomy. Resection of a small portion of the tumour has been followed by resolution

of the remaining portion in a number of cases. Spontaneous retrogression has been reported. When the disease is limited to the gland, removal presents no difficulty; but in cases with extensive extra-thyroidal lesions, operation is difficult and dangerous, and success depends largely on the judgement of the operator in deciding the moment at which to stop before damage is done to the cervical structures. Fatal accidents have occurred where removal has been attempted in the face of severe adhesions. Tracheotomy is not advisable unless the dyspnoea is extremely urgent. If complete extirpation is impossible, partial removal offers the best prospects. The inoperable cases die of suffocation.

The affection passes under various names which express more or less the importance of some clinical or pathological character, viz., 'primary chronic canceriform inflammation' (Tailhefer); 'primary chronic inflammation' (Berry), 'ligneous thyroiditis' of French writers, 'fibrous degeneration' (Ricard), 'struma lymphomatosa' (Hashimoto), 'benign granuloma' (Ewing), and 'chronic productive thyroiditis' (St. George).

The disease seems to have attracted little attention in this country, owing, no doubt, to its rarity. Berry gives a concise account from the clinical side (including 3 cases) in his *Diseases of the Thyroid Gland*, and the pathology is succinctly described in Ewing's *Neoplastic Diseases*. Apart from these there is no comprehensive description, and in the current text-books the disease is mentioned very briefly. We have collected only 23 cases from the literature since 1896, and these we have arranged in tabular form so as to illustrate points of interest in diagnosis, treatment, course, and sequelæ.

In the last few years we have seen 6 cases, the study of which presents several interesting features both clinical and pathological. They form a series illustrating successive phases in the clinical and pathological evolution, and show that the original conception of Riedel's disease must be extended to include the condition described by Hashimoto in 1912 as 'struma lymphomatosa', this being really the early stage of the affection. The histological changes are inadequately described in the literature and not generally recognized. In several cases the condition has been mistaken for malignant disease. We have therefore described the histology in detail. In addition, one of our cases suffered from Addison's disease, and the adrenals showed changes analogous to those in the thyroid. This, we believe, is the first time the association has been recorded.

### PARTICULARS OF CASES.

*Case 1.*—Female, age 36. Admitted to hospital with typical Addison's disease of one year's duration. No history of thyroid disturbance or of enlargement of the gland. She died two months after admission.

**SECTION.**—The thyroid, which had caused no symptoms during life, was moderately and uniformly enlarged and hard. The cut surface was white, opaque, solid-looking, and devoid of colloid. The capsule was not appreciably thickened, and there were no adhesions. Larynx and trachea normal. Both adrenals showed extreme fibrous atrophy, their maximum thickness being 3 mm. The thymus weighed 16 grm., but there was no general lymphatism. The pituitary, pineal, and pancreas showed no special change histologically. There was no evidence of tubercle in any of the organs.

## HISTOLOGY.—

1. *Thyroid*.—Sections from several parts of the gland show the same changes. The capsule is slightly thickened by mature fibrous tissue in which are scattered foci of round cells and islets of atrophied epithelium. The external surface is ill-defined and evidently invading the surrounding adipose tissue. The capsular vessels are normal. The interlobular septa are thickened, but there is no diffuse fibrosis. The most striking feature is the intense round-cell infiltration, which has overrun the gland, replacing the acini in many areas and forming numerous lymph-follicles with large germ-centres (*Fig. 51*). The majority of the cells are lymphocytes, but there are numerous plasma cells and some larger phagocytic cells. The whole are lodged in a delicate reticulum of collagen fibrils and young fibroblasts. There are very few capillary blood-vessels. The lymph-follicles are round and variable in size; the germ-centre, usually about four-fifths the diameter of the follicle, is bounded by a narrow zone of lymphoid cells. Reticulum fibres seem to be absent in the germ-centre, which is mainly composed of large pale reticular cells, loosely arranged and

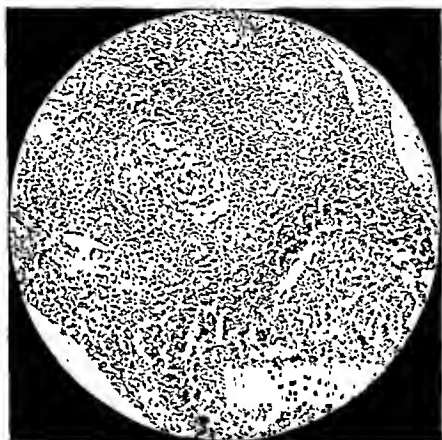


FIG. 51.—Case 1. Thyroid showing early stage with diffuse cellularity. Surviving gland tissue is hypertrophic. No fibrosis. Sector of a lymph-follicle to one side.

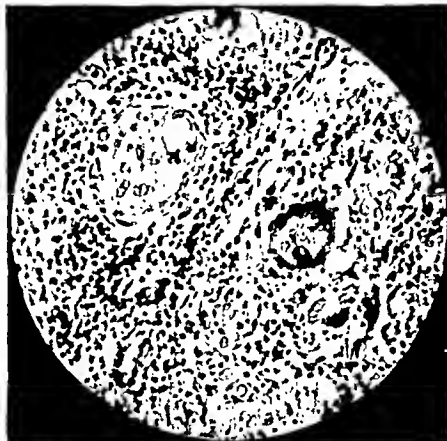
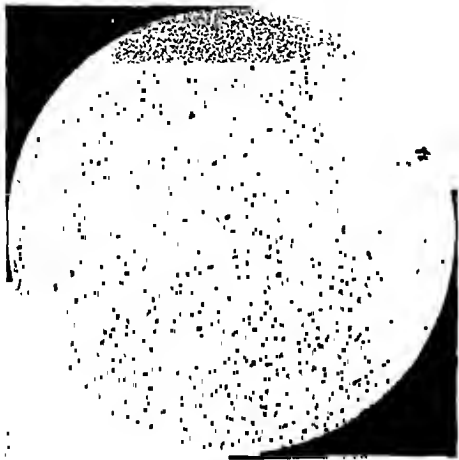


FIG. 52.—Case 1. Thyroid showing diffuse cellularity, solid hypertrophic acini, and two giant cells.

easily washed out of the preparation. These cells are actively phagocytic to the remains of thyroid epithelium and droplets of colloid which are scattered about, and often show mitosis. Everywhere the acini are altered and reduced in number. Occasionally where there is little cellularity the vesicular structure is retained, the epithelium is normal, and colloid is present; but for the most part the colloid has disappeared and the acini are solid. The epithelial cells are much enlarged, the nucleus is prominent and sometimes lobulated, the protoplasm vesiculated (*Fig. 52*). A few cells show mitosis. Sometimes a small lumen is present containing a watery material or, perhaps, a droplet of thick colloid. The appearances clearly indicate increased functional activity. At the same time there is evidence of degeneration in many of the cells: pyknosis, crenation, chromatolysis of the nucleus, and invasion of the cell-body by phagocytes from the surrounding round-cell infiltration. Where the acinus still contains a lumen, the epithelial cells become detached and break down. Frequently a mass of these cells fuse together—often round a drop of colloid as a nucleus—to form a giant cell, which is then invaded by phagocytes and ultimately destroyed (*Fig. 52*). Some of these formations, especially when they present a homogeneous centre of colloid with a peripheral ring of nuclei, closely resemble tubercle giant cells. There is, however, no doubt that they are epithelial in origin.

2. *Adrenals*.—The changes are the same in both organs. Parenchymatous tissue is present only in the central portion of the gland; it is so altered by degenerative change that it is difficult to be sure of its nature, but it appears to be cortical tissue. The cells lie in small groups, isolated by a delicate vascular connective tissue infiltrated with round cells, these being lymphocytes, plasma cells, and some phagocytes. There are a few small lymph-follicles with germ-centres, but these are much less developed than in the thyroid. At the peripheral parts of the gland all trace of adrenal tissue has disappeared, so that at one pole the stroma which contains some

foci of round cells has collapsed, allowing the capsule of the opposite sides to come together. At the other pole the stroma is densely infiltrated with round cells, among which is the debris of adrenal tissue (*Fig. 53*). The capsule of the gland is thickened by a hyaline fibrosis. There is no evidence of tubercle.



*FIG. 53.*—*Case 1.* Adrenal showing similarity of the change to that in the thyroid.

*Case 2.*—Female, age 32; under the care of Mr. G. Grey Turner.

ON ADMISSION.—There is a small painless tumour in the neck which had been present for six months and was getting larger. She had recently suffered from a severe and prolonged furunculosis treated by vaccines, but apart from this there was nothing of note in her history. There is an extremely hard painless nodule in the right lobe. The thyroid itself is not enlarged, and the nodule caused no symptoms. There are no signs of functional disturbance. As the nodule was growing and the patient of nervous

temperament, it was decided to remove it. The nodule was fixed and difficult to enucleate; there was no excessive bleeding.

SECTION.—The nodule was very hard, opaque, and white, and described as 'anæmic fibrosed thyroid tissue'. No clinical opinion was expressed as to its nature. Normal recovery.

She has been seen periodically, and at the present time (two and a half years after operation) is in good health and without recurrence.

HISTOLOGY.—The structure is similar to that of the thyroid in *Case 1*, the only differences being that the capsule of the nodule and the interlobular septa are thicker from fibrosis, normal acini are more numerous, and there is less evidence of hypertrophy of the gland cells.

*Case 3.*—Female, age 55, under the care of Mr. R. J. Willan.

ON ADMISSION.—There was a painless swelling on the right of the neck; first noticed one year ago, when it was the size of a pigeon's egg. She suffers from attacks of nocturnal palpitation, dyspnoea on exertion, and slight dysphagia; no dysphonia. She says she has not lost weight. Her previous history is without note, and she has never had goitre before this. She is a spare woman, without any signs of functional disturbance of the thyroid. The right lobe of the gland forms a bulky, very firm tumour, not fixed to the skin or deeper structures. The lymph-nodes are not enlarged. The rest of the thyroid appears normal. The tumour was thought to be a hard adenomatous goitre.

OPERATION.—Right hemithyroidectomy on the day after admission. The tumour came away easily and with little bleeding. There was some pressure on the trachea. The mass weighed 150 grm.; it was firm, solid, creamy-white, smooth on surface, without fibrosis or thickening of the capsule. It was thought to be sarcoma.

Uneventful recovery. She is now in good health and without recurrence twelve months after operation.

**HISTOLOGY.**—Sections from different parts show the same changes. The appearances, however, are not the same as in the previous cases. There has been great destruction of the acini. The lobular structure of the gland is gone, the whole tissue being invaded by a profuse infiltration of cells which at first suggest a sarcomatous growth. The cellular infiltration is composed of lymphoid cells, plasma cells, and phagocytes lodged in delicate fibrous reticulum, but differs from the other cases in the presence of a great number of cells which appear to be young active fibroblasts. These cells are large, pale and plump, irregularly shaped, and with fine processes. The nucleus is large and vesicular, round, oval, or lobulated. Mitoses are common. There appear to be transitions between these cells and phagocytes. Capillaries and other vessels are scanty. Lymph-follicles are absent. Although fibroblasts are so abundant, the only evidence of fibrosis is the fine diffuse reticulation. Neither the capsule nor the fibrous septa are thickened. The capsular vessels are normal. The surviving acini appear either as colloid-containing vesicles or as solid structures showing both hyperplastic and degenerative changes. There are no giant cells, owing probably to the great destruction of parenchyma. A large amount of disorganized epithelium is strewn about, and this, with the marked activity of the fibroblasts without appreciable fibrosis, indicates recent and severe glandular demolition with early repair. Attached to the exterior of the capsule is some adipose tissue in which are foci of round cells, evidence of early extracapsular extension.

**Case 4.**—Female, age 47, under the care of Dr. Pearson whom she first consulted, nine months prior to admission, for shortness of breath on exertion. He found a uniform, firm, painless enlargement of the thyroid. Three months later the right lobe had become larger and harder. During the next six months the dyspnoea increased, dysphagia, hoarseness, and attacks of aphonia developed, and for these she was admitted under Mr. Collingwood Stewart for operation. The majority of the right lobe was removed. It came away easily and with little bleeding. No fixation to surrounding tissue. It was thought to be malignant, and was not examined histologically. The wound healed per primam, and symptoms were relieved.



Fig. 54.—Case 4. Present state. Enlarged left lobe and isthmus. Right lobe removed two and a half years previously.

Two years later she returned with severe dyspnoea and the history that two months after operation a swelling appeared in front of the neck. A small very hard tumour was found in the isthmus. This was removed with difficulty owing to adhesions. The specimen as received by us measured 5.0 cm. by 3.0 cm., and showed the gross and histological changes of Riedel's struma. The wound healed normally, and dyspnoea was relieved.

She returned three months later, when she was first seen by us, because of swelling of the left lobe with dyspnoea on exertion, dysphagia, and hoarseness. She is a spare pale woman showing no signs of hypo- or hyperthyroidism. There is a history of rheumatic fever in childhood, and there are old scars of tuberculous glands in neck. No goitre in family. The left lobe is enlarged (9.0 cm. by 4.0 cm.), smooth, painless, and feels like cartilage (Fig. 54). The upper margin extends nearly to angle of jaw,



the lower pole is just behind sternoclavicular joint. The tumour moves on deglutition, but is firmly fixed to the trachea; the posterior edge is ill-defined. The overlying skin is not adherent; the lymph-nodes are not palpable. The remains of the isthmus are slightly enlarged and extremely hard. No signs of recurrence on right side. The trachea is pushed to the right. Laryngoscopy (Mr. Frank Wilson) shows left recurrent paresis, fixation of left cord, over-abduction of right cord, reduction of rima to half its normal size, but no evidence of recent or old inflammation. X rays shows no tracheal stenosis, and the mediastinum appears normal. Blood examination: some secondary anæmia. Wassermann, negative (before and after a provocative dose). During the last three months she has been seen periodically. The swelling is slowly increasing, dyspnœa is more marked, and she is having nocturnal attacks. Recently she has had a course of N.A.B., as it was thought that this might prove beneficial and so avoid removal of the tumour. It has had no effect.

**HISTOLOGY.**—Portion removed at second operation. This is similar to Cases 1 and 2, but in a more advanced stage. The capsule is much thickened by hyaline fibrosis; it contains foci of round cells and atrophied acini, but no extracapsular

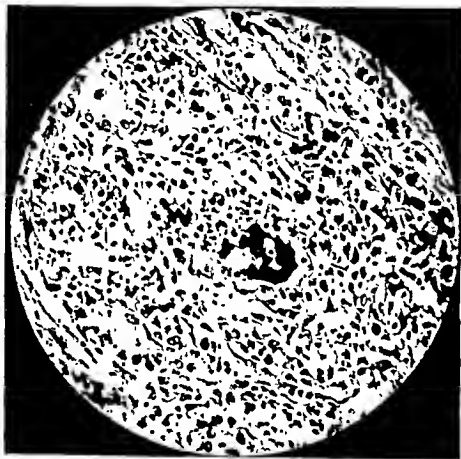


FIG. 55.—Case 4. Thyroid. Area of demolition showing early fibrosis. Giant cell degenerating.

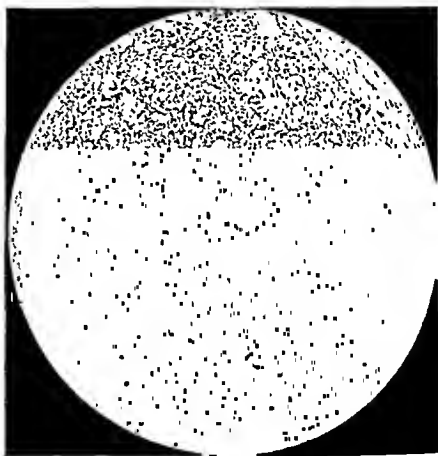


FIG. 56.—Case 4. Thyroid showing hyaline fibrosis at periphery of field.

structures. The capsular arteries are thickened. Running throughout the gland are broad interlacing bands of hyaline fibrous tissue in which are incarcerated remains of acini, foci of round cells, and atrophied lymph-follicles. At the edges of these bands the earlier stages of fibrosis can be seen, where active fibroblasts and collagen fibrils are being laid down among the disintegrated acini (Fig. 55). The round-cell infiltration is not so marked, as it is being replaced by fibrous tissue (Fig. 56). In the lymph-follicles the cells of the germ-centre are less active and lie in a distinct fibrous reticulum. When the follicle is eventually overcome by the advancing fibrosis it undergoes atrophy, but the large pale reticular cells persist for some time, and might be mistaken for groups of malignant cells. In the surviving acini the epithelium is more hypertrophic than in the other cases, and in several places forms small solid adenomatous-like masses in which the cells have large, sometimes lobulated, nuclei. These areas are practically free of round-cell infiltration, and are really foci of compensatory regeneration.

**Case 5.**—Female, age 58. admitted to hospital under Mr. John Clay with intense dyspnœa.

**HISTORY.**—A painless swelling appeared in the neck about four years prior to admission. It was first noticed by her friends, and she is unable to say whether it began diffusely or locally. About a year and a half later she had some dyspnœa, which in the last year has become worse, causing suffocative attacks at night. She has never had dysphagia, but dysphonia developed shortly after swelling appeared, and at times there is aphonia. The tumour has varied in size from time to time, but on the whole was slowly progressive until a year and a half ago, when it enlarged more rapidly. No goitre in family. Medical history without note.

**PRESENT CONDITION.**—The patient is of fair nutrition and shows no signs of functional thyroid disturbance. Blood-pressure, 218 mm. Hg. Urine contains a trace of albumin. Blood examination, nil. Wassermann, negative. Thyroid is diffusely enlarged and described as being 'as hard as stone'. Skin not adherent. Lymph-nodes not enlarged. The intense dyspnœa demands immediate relief. Clinical diagnosis, malignant struma.

**OPERATION.**—Total thyroidectomy under local anæsthesia. Gland removed with great difficulty, as it was very adherent to the trachea and surrounding tissues. No undue bleeding. Wound healed per primam. Thyroid tablets given. She has been under observation for the last two and a quarter years without showing any recurrence, and continues in fairly good health.

The thyroid weighs 118 grm.; it is uniformly enlarged and extremely hard. The surface is slightly nodular and the capsule thickened. On section it shows the usual appearances and fibrosis is easily seen.

**HISTOLOGY.**—This is essentially the same as *Case 4*, but there is less gland tissue and more fibrosis. The capsule is much thickened by hyaline fibrosis, and there is definite evidence of extracapsular infiltration as shown by atrophic striped muscle-fibres and islets of adipose tissue incarcerated in the fibrous tissue. Foci of round cells exist far beyond the confines of the original capsule. Capsular arteries are thickened. (*Fig. 57.*)

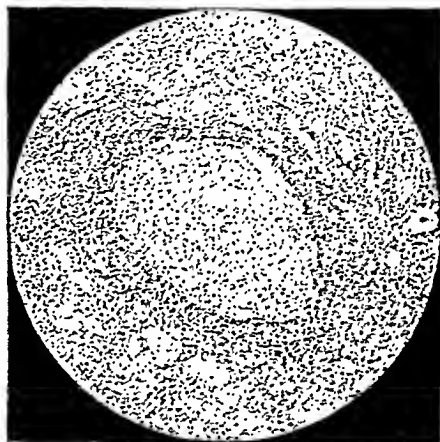
**Case 6.**—Female, age 46, admitted under Mr. T. A. Hindmarsh with dyspnœa and painless swelling in neck. About ten months prior to admission her relatives first noticed the swelling, which has increased slowly. Six months later hoarseness and dyspnœa with suffocative attacks developed. No dysphagia. Previous history without note. No goitre in family. Relief sought for dyspnœa.

**ON ADMISSION.**—She is a well-nourished woman with no signs of myxœdema or hyperthyroidism. Thyroid diffusely enlarged, rather nodular, and very hard; it moves on swallowing; posterior margins definable. Skin not adherent. Glands not palpable. Blood examination, nil. Wassermann, negative (before and after provocative dose). Clinical diagnosis, cancer of thyroid.

**OPERATION.**—Total thyroidectomy day after admission. The gland was very adherent to the trachea and was freed with great difficulty. No undue bleeding.

The organ weighs 190 grm.; the appearances are similar to gland from *Case 5*, but the surface is definitely nodular and on section the fibrosis is more obvious (*Fig. 58*). Uneventful recovery. Dyspnœa cured. Thyroid tablets given. Patient is in good health and without recurrence three years after operation.

**HISTOLOGY.**—Similar to *Cases 4* and *5*, but it is the most advanced case in the series as regards the changes within the capsule. Large areas of the gland are



**FIG. 57.**—*Case 5.* Thyroid. Lymph-follicle with large germ-centre. Diffuse cellularity and solid acini.

replaced by a dense hyaline fibrous tissue (*Fig. 59*) in which are sometimes the remains of acini and lymph-follicles. Elsewhere there is the usual round-cell infiltration and lymph-follicle formation. The surviving acini are solid, the epithelial cells being markedly hypertrophied. In addition there are several solid adenomatous-like nodules of compensatory regeneration in which mitoses are seen. The signs of increased functional activity are more striking in this case than in any of the others. Extracapsular extension is shown by the inclusion of muscle and adipose tissue in the very hyaline fibrosis which has thickened the capsule. The capsular vessels are also thickened.



FIG. 58.—Case 6. Thyroid weighing 190 grm. removed by operation. The left lobe has been cut to show the solid, white, opaque structure intersected by fibrous bands.

The changes in this gland are instructive because the original histological diagnosis was carcinoma. We believe that this opinion was based on the presence of the adenomatous hyperplasia, the solid acini, and the hypertrophic cells with their large and rather irregularly-shaped nuclei.

### SUMMARY OF CASES.

1. Six cases of Riedel's struma, all in females between the ages of 32 and 58 years.

2. Three of these (Nos. 1, 2, 3) are in an early stage, the disease being confined to the interior of the gland, which shows a peculiar round-cell infiltration without appreciable fibrosis. Two (Nos. 1 and 2) had no symptoms; the other (No. 3), with an unusually bulky tumour, had symptoms of compression.

3. Three cases (Nos. 4, 5, 6) are relatively advanced, the process having spread outside the thyroid, but not sufficiently far to prevent removal. In each case there is fibrosis in the gland. Compression symptoms present in all.

4. In 4 out of 5 cases the clinical diagnosis was malignant disease, and in one the pathological diagnosis was carcinoma.

5. Two cases with local enlargement were cured by removal, which was done without difficulty, but in one case with involvement of the whole gland partial removal has not cured. Total thyroidectomy cured two cases with general enlargement.

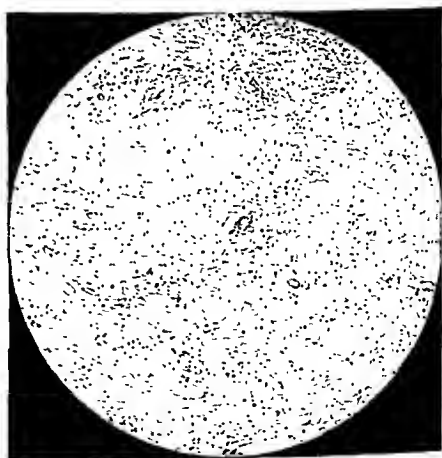


FIG. 59.—Case 6. Illustrating advanced sclerosis. No trace of thyroid tissue.

## DISCUSSION.

The study and interpretation of the histogenesis in Riedel's struma are important in pathological diagnosis and in explaining certain clinical features of the disease.

The early stage is characterized by a diffuse round-cell infiltration and the formation of numerous lymph-follicles with prominent germ-centres. The cells of the infiltration are lymphocytes, plasma cells, and some phagocytes, all lodged in a delicate reticulum of collagen fibrils and fibroblasts poorly supplied with blood. At first there is little reduction in the amount of parenchyma. A variable number of the acini are filled with colloid and lined by fairly normal cells, but in the majority the vesicular structure is lost. The colloid has disappeared; the cells are hypertrophied, and show all the signs of increased functional activity. Cellular degeneration proceeds simultaneously with hyperplasia, and the debris is removed by phagocytosis. The altered cells on detachment from the acinar wall often fuse together, perhaps round a droplet of colloid, to form giant cells which are eventually removed. These formations have rarely been described, but are mentioned by Hashimoto and Monod. They have been mistaken for tubercle giant cells (Wilke). Numerous in the earlier stages of the disease, they disappear as fibrosis advances. With progressive glandular destruction fibrosis appears, and all stages can be traced in its formation. The sclerous tissue early undergoes hyaline change, and gradually replaces the atrophic acini, the round-cell infiltration, and the lymph-follicles. The germ-centres of the latter persist for some time, and their inclusion in the hyaline fibrous strands produces a picture very like carcinoma. Epithelial hypertrophy is now more evident, and in addition adenomatous foci of compensatory regeneration develop. They form rounded nodules sometimes visible to the naked eye in histological sections. Vesiculation is absent, the mass being formed of solid acini of large cells with prominent, sometimes lobulated, nuclei occasionally in mitosis. There is no cellular infiltration in these foci, and the stroma is similar to normal thyroid. The general structure and arrangement of the nodules leave no doubt as to their nature, and is in conformity with the fact that glandular hyperplasia is readily induced in the thyroid. Hyperplasia seems to have been observed by Silatschek, though he gave it another interpretation, and it is briefly mentioned by Monod, and by Delore and Alamartine. It probably accounts for the pathological diagnosis of carcinoma (Silatschek, Balfour). The change is similar to the compensatory regeneration which takes place in cirrhosis of the liver, and which sometimes passes over into carcinoma. From analogy it is reasonable to imagine that such an event might occur in the hyperplastic nodules of Riedel's struma, but up to the present we have no evidence of this transformation. The absence of myxœdema even with a very cirrhotic gland can only be explained by the glandular hyperplasia, though no doubt in cases in which only a part of the gland is diseased the remainder provides sufficient secretion. Indeed, the glands in myxœdema and in advanced Riedel's struma are very similar, the only essential difference being the glandular hyperplasia in the latter. The remarkable hardness of the gland cannot be explained by fibrosis alone, as the highly cellular tissue

of the early stage produces the same physical change. In the cases described by Riedel and others no mention is made of diffuse cellular infiltration and lymph-follicles. The gland was very cirrhotic and contained foci of round cells and a few vesicles. But in 1912 Hashimoto described four cases of uniform enlargement without symptoms in which the histology was identical with our early cases (Nos. 1 and 2). He considered them as forming a distinct entity—'struma lymphomatosa'—unrelated to Riedel's disease. Ewing, however, has pointed out that Hashimoto and Riedel were describing the early and late stages of the same process, and our series entirely confirms this conclusion.

The pathogenesis of the early stage requires interpretation. The infiltration with its numerous lymph-follicles certainly resembles lymphoid tissue

very closely. But there are several difficulties in accepting the view that the tissue is pure lymphoid overgrowth. In the first place, lymph-follicles are known to develop where there is chronic inflammation, viz., Fallopian tube, gall-bladder, endometrium, etc. Recently we have seen an example of this in a man of 29 who had a painless tumour of about 1½ in. diameter, as hard as stone, just beneath the right lower jaw. The overlying skin was red, hot, and adherent. X rays showed no calculus. At operation a drop of pus was found. Microscopically it proved to be salivary gland. There was diffuse fibrosis and round-cell infiltration; much glandular atrophy with some polymorph leucocytes in the ducts. The whole tissue was studded with numerous lymph-follicles containing

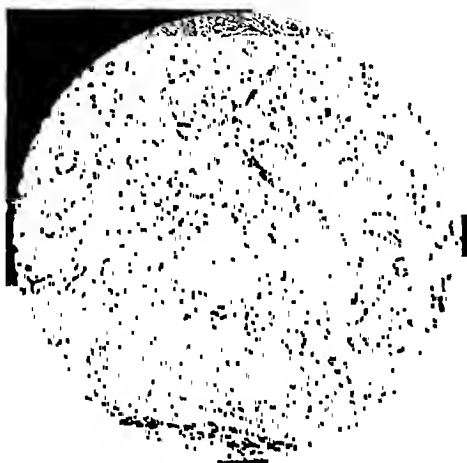


FIG. 60.—Salivary gland in chronic inflammation. The diffuse cellularity, glandular atrophy, lymph-follicles, and fibrosis are similar to the structure of the thyroid in Riedel's struma.

large germ-centres (*Fig. 60*). The condition was clearly inflammatory in origin, yet very similar in structure to Riedel's struma.

Moreover, lymphoid foci may develop in various affections of the thyroid, but especially in Graves' disease, a condition in which the gland-cells are active. In Riedel's struma the gland-cells are also active, and the presence of lymph-follicles is probably to be associated with this state. Their number and size tend, however, to divert attention from the other changes which, we believe, are essentially a chronic inflammatory process. This is borne out by one of our cases (No. 3) in which there were no lymph-follicles. Here there seems to have been rapid and extensive destruction of the parenchyma and the formation of a definitely granulomatous tissue, which might be mistaken for sarcoma. Plasma cells were present in all our cases, and in much larger numbers than are found in lymphoid tissue. Their excess speaks for an inflammatory process. Lastly, the extension of the process outside the capsule, spontaneous recession of the tumour, and retrogression after imperfect

removal are all clinical features more in favour of an inflammatory process than an involutionary change followed by simple lymphoid overgrowth. Thickening of the arteries of the gland is common, particularly in advanced cases. Some have thought this may be due to syphilis, but there is nothing to support the idea. Either the age of the patient or the sclerotic condition of the gland is sufficient explanation.

**Extracapsular Extension.**—This develops early, and is present histologically before it is clinically recognizable. In none of our cases was it bad enough to prevent operation. It is described as a tough, leathery, white tissue, prone to bleed. Microscopically it is densely fibrotic or a fibrocellular tissue with round-cell infiltration. In moderately advanced cases it forms adhesions to the vessels, trachea, nerves, and muscles, and shows a marked capacity to infiltrate the tissues, so that at operation it is difficult or impossible to recognize the cervical structures, which are bound in a dense sclerous overgrowth. It may invade the trachea (Riedel), gullet (Ricard), and carotids (Delore and Alamartine). Remarkable extensions have been described in more advanced cases; the infiltration may pass up to the base of the skull (Tailhefer), or more commonly down into the mediastinum (Monod, Bohan, Ricard, Berry), enveloping the trachea, bronchi, and great vessels. Strange to say, the skin is rarely involved, though this has been reported by Monod, and in Berry's case led to ulceration. During life there is cyanosis and intense dyspnoea; the neck is swollen, very indurated, and the normal topography is lost. Cases of this type usually prove fatal whether left to Nature or subjected to operation. The extent of the lesions has been confirmed at autopsy. The infiltrative character of the tissue is so remarkable that in Berry's case the condition was described as an 'infiltrating fibroma', yet microscopically there was no malignancy. For a summary of the various difficulties and accidents connected with such cases the reader is referred to *Table I*.

**Lesions in Larynx and Trachea.**—Compression and parietic lesions are common; but it has often been remarked that the dyspnoea may be out of proportion to the size and site of the tumour. St. George's case is instructive on this point. The patient died of erysipelas following tracheotomy for severe dyspnoea. At autopsy the thyroid was not enlarged, but adherent to, and constricting, the trachea. Microscopically it was very fibrous. This case suggests that fibrous contraction in the gland itself may sometimes be the cause of dyspnoea. Inflammatory lesions have been recorded in three cases. One of Riedel's patients returned twelve years after operation cured of his struma, but with severe dyspnoea from subglottic stenosis 17 cm. from the teeth. Silatschek describes an acute tracheitis and narrowing of the first four tracheal rings. Monod found ulceration below the glottis and at the tracheal bifurcation in a case which died. He concludes that Riedel's struma is not a primary disease of the thyroid at all, but is secondary to a tracheal lesion which is probably syphilitic. There is nothing to support Monod's view. If it were true, inflammatory lesions should be common, but the larynx and trachea are usually normal, and this has been confirmed post mortem in one of our cases and by other writers. The cases of Riedel, Silatschek, and Monod all had extracapsular lesions which more probably invaded the trachea secondarily, in the same way as they are known to attack other structures.

Table I.—SUMMARY OF 23 CASES OF

AUTHOR	SEX AND AGE	DURATION AND SITE	SYMPTOMS	WASSERMANN	EXTRACAPSULAR LESIONS
Riedel, 1883	M. 42	6 months, diffuse ..	Dyspnœa	O	Adherent to vessels, both sides ..
Riedel, 1896	F. 23	1 year, diffuse ..	Dyspnœa, dysphagia	O	Adherent to vessels and recurrent ..
Riedel, 1896	M. 29	6 weeks. Right lobe first, then rest of gland .. ..	Dyspnœa	O	Adherent to muscles, vessels, and nerves
Berry, 1883. (Bowlby's case)	F. 42	3 years, diffuse ..	Dyspnœa, dysphagia, dysphonia ..	O	Adhesions to muscles, vessels, nerves, gullet, trachea. Extension to mediastinum
Berry, 1898	F. 40	5 weeks. Right lobe	'Shooting pains' in neck .. ..	O	None
Berry, 1899	F. 63	Symptomless goitre for many years. Diffuse .. ..	Dyspnœa for two months .. ..	O	Adhesion to trachea, left carotid, and ..
Berry, 1921	F. 46	2 months. Hazel-nut nodule in right lobe	Some pain ..	O	None
Tailhefer, 1898	M. 30	3 months. Left lobe	Aphonia	O	Adherent to left carotid sheath and extension to base of skull
Ricard, 1901	M. ?	Left lobe .. ..	Dysphonia, dyspnœa, dysphagia ..	O	Adherent to muscles, vessels, vagus, trachea, gullet; behind clavicle ..
Hashimoto, 1905	F. 55	4 weeks, diffuse ..	Slight dysphagia and dysphonia ..	O	None
Hashimoto, 1907	F. 61	7 months, diffuse ..	None	O	None
Hashimoto, 1907	F. 45	3 weeks, diffuse ..	None	O	Fixed at its base
Hashimoto, 1909	F. 40	6 weeks, diffuse ..	Slight dysphonia ..	Negative	None
St. George	F. 35	4 months, diffuse ..	Dyspnœa	O	Adherent to trachea
St. George, 1905	M. 25	3 months. Right lobe .. ..	Dyspnœa, dysphagia	O	Adherent to trachea, gullet, recurrent nerve; behind clavicle .. ..

O = No statement.

CASE COLLECTED FROM THE LITERATURE.

TREATMENT	CLINICAL DIAGNOSIS	PATHOLOGICAL DIAGNOSIS	RESULT
Bit-sized piece removed .. ..	Malignant	Chronic inflammation .. ..	Tumour receded. Dyspnœa gone in 6 months. Died 15 months after operation from nephritis
.. .. removed ..	Malignant	Chronic inflammation .. ..	Dyspnœa relieved. Died suddenly 2 months later from (?) embolism
.. .. piece removed; no f. 6 months later her small piece removed. Dyspnœa improved .. ..	Chronic inflammatory ..	Fibrosarcoma	Returned 12 years later with dyspnœa from subglottic stenosis. No trace of goitre
.. .. resection of isthmus. Tracheotomy .. ..	Malignant	Chronic inflammation .. ..	Died shortly after tracheotomy. Post-mortem
.. .. right lobe removed ..	? Malignant	Chronic inflammation .. ..	Alive and well 2 years later
.. .. resectable. Tracheotomy	? Inflammatory ? Malignant	Chronic inflammation .. ..	Death from septic pneumonia 6 days later. Post-mortem
.. .. right lobe removed ..	? Cyst ? Malignant	Chronic inflammation .. ..	Cured
.. .. 11 bit removed ..	Cancer	Chronic inflammation .. ..	Secondary hæmorrhage from carotid. Hemiplegia. Tumour had gone about a year later
.. .. removed piecemeal. Vocal cords injured	Cancer	Fibrous degeneration .. ..	Recovery
.. .. complete removal ..	Struma fibrosa	Struma lymphomatosa .. ..	Well 2 years later
.. .. complete removal ..	Parenchymatous goitre	Struma lymphomatosa .. ..	Well 4 years later
.. .. complete removal ..	Struma fibrosa	Struma lymphomatosa .. ..	Well 4 years later
.. .. complete removal ..	Malignant	Struma lymphomatosa .. ..	Well 2 years later
.. .. tracheotomy	O	Chronic inflammation .. ..	Died on 5th day of erysipelas and hæmorrhage. Post-mortem
.. .. removal of right lobe	O	Chronic inflammation .. ..	Alive and well 4 years later

[Continued on next page



Table I.—SUMMARY OF 23 CASES OF

AUTHOR	SEX AND AGE	DURATION AND SITE	SYMPTOMS	WASSERMANN	EXTRACAPSULAR
St. George, 1922	M. 31 (Author himself)	5 months, diffuse, but especially right lobe .. ..	Dyspnœa, dysphagia	Negative	Right lobe adherent to muscles and
Silatschek, 1908	M. 32	1½ years. Right lobe, then rest of gland	Periodic aponia, then dyspnœa. Gland varied in size	Negative	Adherent to surrounding parts
Spannaus, 1909	M. 32	Symptomless goitre for 7 years ..	Dyspnœa and dysphonia last 6 months	Negative	Adherent to vagus, tracheal gullet
Delore and Alamartine, 1910	M. 39	5 months. Right lobe .. ..	Dysphonia, dysphagia	O	Adherent to vessels, muscles, recurrent Carotid and softened
Poncet and Leriche, 1912	M. 60	6 months. Isthmus, and then whole gland .. ..	Dyspnœa	O	Gland fixed
Balfour, 1918	F. ?	Diffuse	Tracheal pressure	O	Gland fixed
Monod, 1921	F. 30	5 months. Left lobe, then rest of gland	Dysphagia, dyspnœa, dysphonia ..	Negative	Diffuse infiltration of neck and upper part of sternum
Bohan, 1924	F. 38	4 months, diffuse ..	Dyspnœa. Cough. Dysphagia. Pain in neck .. ..	Negative	Adherent to Mediastinal by X-ray

O = No statement.

**Frequency.**—The disease is rare. We have collected only 23 cases from the literature (1896–1924). St. George states on the authority of Pemberton that 48 cases were seen in 10,500 thyroidectomies in the Mayo Clinic. Riedel saw 3 in 1064 goitres. Ewing (private communication to one of us) has had 8 cases. The last 150 thyroids examined in our department did not yield a case. Nevertheless the condition is certainly not as rare as these figures suggest. A more critical examination of cases of ‘inoperable cancer’ and ‘cured cancer’ would, doubtless, swell the number of Riedel’s struma. Spontaneous recession may account for a few cases. The histology has been mistaken for cancer or sarcoma. Indeed, there are few diseases in which the clinician, the surgeon, and the pathologist can all err in making a diagnosis of malignancy; yet this happened in Balfour’s case—time alone proving the true nature of the disease.

**Sex and Age.**—In our 23 collected cases 10 were males. The youngest was 23 and the oldest 63 years. Two-thirds of the cases occur between the second and fourth decades. There are verbal statements that the disease

THE CASES COLLECTED FROM THE LITERATURE—*continued.*

TREATMENT	CLINICAL DIAGNOSIS	PATHOLOGICAL DIAGNOSIS	RESULT
lobe removed	Calcified adenoma ..	Chronic inflammation .. ..	Alive and well 1 year later
.. for diagnosis ..	Malignant	Cancer, then chronic inflammation .. ..	Tumour disappeared entirely some months after operation
.. of left lobe removed .. ..	O	Chronic inflammation .. ..	Tumour disappeared 2 months after operation
.. of right lobe	Malignant	Chronic inflammation .. ..	Died of hæmorrhage from carotid 4 days after operation
.. us divided ..	O	Cirrhosis	Thyroid normal in 1 month
.. movable .. ..	Cancer	Cancer	Alive and well 7 years later. Gland receded
.. sy. N.A.B. course not cure. Tracheotomy .. ..	Malignant, then cellulitis ..	Riedel's disease..	Died some weeks after tracheotomy. Post-mortem
.. us and right lobe removed. No improvement. Tracheotomy ..	Malignant or acute thyroiditis .. ..	Chronic inflammation .. ..	Two months after tracheotomy tumour had disappeared. Attributed by author to removal of septic teeth

may develop in childhood (Cordua, Riedel), but we have found no description of a case.

**Etiology.**—This is unknown. Syphilis and tubercle can be excluded with certainty. In 5 of our cases examined for tubercle bacilli, spirochaetes, and the common bacteria, the sections were negative. Animal inoculation is negative (Poncet and Leriche). The Wassermann was negative in all cases in which it was done, including 3 of our own. Bohan thinks dental infection may be a factor, because the tumour disappeared in one case after removal of three teeth with apical abscesses; but this may have been due just as well to spontaneous retrogression. We have been unable to collect any evidence that the condition is an involution following a previous goitre. In a few cases there has been a history of goitre or Graves' disease in other members of the family. We believe that the disease is a chronic inflammatory process of a granulomatous nature, and this view receives ample support from its clinical characters and the anatomical changes in the gland.

As to the relation, if any, between Riedel's struma and fibrous atrophy

of the adrenals, we are unable to express an opinion, except to say that the anatomical changes are strikingly similar. That the alterations in the thyroid in our case are not the result of some disturbance in the gland following adrenal disease seems to be proved by Huebschmann's observation that in fibroid atrophy of the adrenals the thyroid is normal. At any rate our case suggests the possibility that the factor responsible for Riedel's struma may also attack other members of the endocrine glands.

Our special thanks are due to Mr. J. Clay, Mr. T. A. Hindmarsh, Dr. Pearson, Mr. Collingwood Stewart, Mr. Grey Turner, and Mr. R. J. Willan, whose courtesy and help have made it possible to record the clinical histories of their cases.

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### THREE CASES OF ANEURYSM OF THE AORTA TREATED BY WIRING.

[THESE cases appear to be worthy of publication, as they deal with a method of treatment which is still upon its trial; they illustrate some of the difficulties of the operation, and they are a continuation of a series which has already been published in the JOURNAL (Vol. ix, pp. 27-36).—ED. B.J.S.]

#### I. ANEURYSM OF THE ABDOMINAL AORTA.

By G. H. COLT, ABERDEEN.

D. R., 78, a retired marine engineer, was admitted to the Aberdeen Royal Infirmary under my care on Dec. 14, 1923, and died Feb. 13, 1924. For about fourteen months the patient had suffered from pain in the back. The onset had been gradual. The pain was sometimes of a gnawing character and was fairly constant. He was able to ease it by lying face downwards. For five weeks before admission he had suffered from intermittent attacks of cramp in the abdomen accompanied by sickness and vomiting, and during this time his bowels became increasingly constipated. The pain was most marked about half an hour after food, and was related more to the quantity than to the kind of food taken. During the last year he had become progressively thinner, and for about a month had noticed a swelling in the middle of his abdomen. In the course of his profession the patient had visited many lands. He denied having had venereal disease, and was the father of a healthy grown-up family of four.

ON EXAMINATION.—The patient was a spare and rather feeble man, but in general remarkably healthy. The tongue was dry and furred and the appetite poor. The respiratory excursions were equal on the two sides. The percussion note at both bases was impaired and the breath-sounds were subdued. There were no moist sounds. My colleague, Dr. W. F. Croll, who saw the patient with me, reported: Pulse 72, occasional intermissions, pulse full between beats. Systolic blood-pressure 160 mm. Hg, diastolic blood-pressure 140 mm. Hg, pulse-pressure 20 mm. Hg, vessel-walls slightly degenerated. The low pulse-pressure suggests myocarditis. Extended open impulse. Apex in 5th interspace about 4 in. from mid-sternum. Impulse rather thrashing in character. No enlargement of right side of heart made out. Sounds good all over. Slight accentuation of second aortic sound. A faint murmur accompanies first sound at all areas. Electrocardiogram shows ventricular premature beats arising in the right ventricle, otherwise the electrocardiogram is perfectly normal in every respect. The femoral pulse is quite strong. The delay between the apex impulse and the femoral pulse is not increased beyond the normal 0.24 secs. The Wassermann reaction was negative.

The abdomen showed visible pulsation in the region of the umbilicus, and on palpation a rounded swelling of about 5 in. diameter with the umbilicus as

its mid surface-point was made out. The pulsation on some days was expansile and on others was not definitely expansile. It remained unaltered when the patient was in the knee-elbow position. A definite soft systolic bruit was audible, sometimes to the left and sometimes to the right of the umbilicus. It did not disappear or change in character when the patient sat up; but as the bruit was faint, Corrigan's sign could not be said to be negative. No thrill was made out. The X-ray examination in the lateral position showed no erosion of the vertebræ. The anterior rounded body of the mass was well defined. The opaque-meal examination showed that the pyloric tube was displaced upwards and to the right, and that the outline of the stomach was irregular. There was no definite evidence of delay or of a growth causing obstruction.

The case was diagnosed as one of saccular abdominal aneurysm arising from the region of the cœliac axis, but situated rather lower than is usual in such a case. The absence of a thrill and only the occasional presence of a soft bruit rendered it probable that the opening into the aorta was not a small one. According to previous observations, confirmed post mortem, the harsher the bruit and the greater the thrill, the more nearly circular and (within limits) the smaller is the opening into the aorta, provided the contents of the sac are mostly liquid blood. It is under these conditions that a negative Corrigan's sign would render the diagnosis doubtful. There was no sign of aneurysm elsewhere.

The patient rested in bed and had a light but nutritious diet. Fluids were not restricted. His general condition improved a little. A fortnight after admission there was a sudden increase in the size of the mass, and the pulsation became more marked. There was no sign of hæmorrhage, and the symptoms decreased again in five days. This attack was not repeated, although it is not unusual to see them occur at intervals of two to three weeks. The probable explanation lies in a leakage of blood taking place between the sac and the clot lining the walls. There were intermittent attacks of abdominal pain from which the patient obtained some relief by changing his posture, but his rest was much disturbed and his general condition began to get worse.

OPERATION.—On Feb. 5, 1924, under general anæsthesia, the abdomen was opened through a right paracentral incision and the diagnosis verified. The aneurysmal sac projected from the aorta through and under the transverse mesocolon. The third part of the duodenum was adherent to its upper anterior surface. The lateral parts of the sac were harder than the anterior part, where the sac felt thin. A purse-string suture of linen thread was placed here, the part was punctured, and a wisp of wires  $3\frac{1}{2}$  in. long, having a total clotting surface of  $2\frac{1}{2}$  square in., was gently inserted. The usual precautions were taken, viz., to see blood spurting from the cannula and to feel the lie of the clot in the sac so that the arrangement and expansion of the wires could be to some extent controlled. A small amount of clot, felt with the cannula, lay on each side. Most of the interior of the sac appeared to contain liquid blood. The cannula was withdrawn until the orifice was estimated to be about an inch inside the sac at the time the wisp was inserted. The purse-string suture was tied and the abdomen closed. Excluding the

anæsthetic, the whole operation took twenty-five minutes, and could have been done more rapidly.

The patient had little pain or discomfort after the operation, and made good progress for a week. On Feb. 12, at 9 a.m., he began to vomit and continued to vomit duodenal contents copiously all day. The vomiting stopped during the night, but at 7 a.m. on the next day he suddenly turned grey, and died in about two minutes, without showing any distress or cyanosis, seven days and twenty hours after the operation.

POST-MORTEM EXAMINATION.—A partial post-mortem was allowed. There was acute dilatation of the stomach and duodenum as far as the third portion, where it was adherent to the sac of the aneurysm. The sac arose from the aorta by an irregular opening measuring  $2\frac{1}{4}$  in. vertically and  $1\frac{3}{4}$  in. horizontally. Its axis was situated obliquely, projecting forwards, upwards, and to the left, and measured  $4\frac{1}{2}$  in. The horizontal measurement was  $3\frac{1}{2}$  in. and the antero-posterior  $2\frac{1}{4}$  in. There had been no rupture of the sac, no formation of dissecting aneurysm, and no leakage at the site of puncture. The sac was filled with dense firm clot, adherent to the walls, but for the most part of recent appearance. That on the left anterior part was  $\frac{1}{2}$  in. thick, red and partly laminated. The clot near the aorta was flaky, and some of it had been separated before I saw the specimen, but otherwise the whole aneurysm was full of dense, firm, even clot in which the wisp lay expanded equally. The ends of the wires for about half an inch were free of clot and lay close to the opening into the aorta. On removing some of the clot with a scalpel from between the wires the section was found to be firm and pale, and the clot could only be removed by cutting it. Microscopically, it showed fibrin, but no organization. The walls of the aorta and iliac vessels showed atheroma.

It was unfortunately my lot to be away for a day at the time the dilatation of the stomach occurred and when the post-mortem examination was made. Stomach lavage or an anterior gastro-enterostomy might possibly have saved this patient. By the time I saw the specimen the surrounding parts had been removed, including the bifurcation of the aorta, and the details of the arrangement of the arteries and veins with respect to the sac could not be ascertained. (It is better to harden these specimens *in situ* and then examine them by cutting a window or windows before proceeding to a more detailed preparation of the specimen. They should not be cut in half, as the wires become displaced.) Some of the flaky portion of the clot near the opening into the aorta had also got rubbed off. Apparently there was no embolus and no thrombosis of any of the mesenteric vessels. It is difficult to avoid the conclusion that, had this patient survived the acute dilatation of the stomach, he would have been cured, but whether the large opening into the aorta would have shed emboli or not it is difficult to say. It is clear from the way in which the wires are arranged that the major part of the contents of the sac was liquid when the wires were inserted, and that in approximately eight days nearly the whole of the contents had become solid, and in due course would have become organized.

In most of the cases of natural cure—a subject I hope to consider somewhat exhaustively at a future time in connection with the clinical duration of the disease—the usual condition found in a saccular aneurysm is that

the solid mass of laminæ shows a concave, smooth surface continued close up to the margin of the orifice and sometimes extending into the aorta. The great mass of evidence, notably in internal arteriovenous aneurysm, is against the occurrence of the detachment of organized clot.

Since the last summary of results of the 16 cases which appeared in the BRITISH JOURNAL OF SURGERY in 1921<sup>1</sup>, we have now to add five more (Nos. 17 to 21) as mentioned below. General Sir M. P. Holt has fortunately called to mind two cases which had been missed. One of these, a case of subclavian aneurysm, in which the patient died five years later from hæmorrhage, will not be included here; the other is No. 17 below. In a third, a male, F. E. N., 49, a boiler-scraper, with a strongly positive Wassermann reaction, there was a saecular aneurysm of the ascending arch the size of a large orange; death occurred from dyspnoea twenty-eight days after operation, and very little ante-mortem clot was found round the wires. The case has not been published, but from the notes I have seen it appears that death was not hastened by the operation. The other three cases are recorded in this number of the JOURNAL, and I am greatly indebted to the authors for having allowed me to see their manuscripts so that the summary could be brought up to date.

17. Male (abdominal), died of peritonitis 7 days after operation (Lentaigne).
18. Female (ascending aorta), died  $8\frac{1}{2}$  months after first operation (Wakeley).
19. Male (abdominal), died 8 days after operation (Colt).
20. Male F. E. N., 49 (ascending arch), died 28 days after operation.
21. Female (abdominal), died 6 weeks after first and 12 days after second operation (Marshall).

In the absence of any authoritative statement of the clinical duration of aneurysm it is difficult to assess the value of any particular method of treatment. While America holds the world's records for thoracic aneurysm treated by wiring and electrolysis, Dublin holds the record for abdominal aneurysm treated by wiring alone. The medical profession in Dublin in the past have added so greatly to our knowledge of the disease that it is most fitting that Sir William Wheeler's first case, a male (T.), age 38, with a positive Wassermann at the time of operation on Aug. 30, 1910, should be alive and working in a brewery to-day, 14 years and 6 months later. His second case, a male of 30, lived in good health and died 4 years and 8 months after operation from leakage of a secondary dilatation; the sac post mortem was solid (King-Fretz). Lawson's patient lived  $10\frac{1}{2}$  years; no details could be ascertained of the pathological state. Up to the present time, in the remaining eleven cases of abdominal aneurysm, one patient lived a year, one some months, one six weeks, and the other eight died within nine days of the operation, *at least three of them from acute dilatation of the stomach*. Some of the others may have died of this, but the accounts are not complete. The three cases were Wheeler's third case, Marshall's, and my own, and it is clear that the condition arises either as a result of the change occurring in a consolidating sac or as a result of some nervous or vascular change induced by the trauma. In Marshall's case the distention was always threatening, but never excessive until twelve hours before death.

Box and Cuthbert Wallace,<sup>2</sup> reviewing 18 fatal cases, say: "The actual

exciting agent is obscure. The condition has appeared to follow or be associated with trauma, surgical operations, possibly the administration of chloroform, lung disease, empyema, retroperitoneal suppuration, and, especially, acute infective fevers and other febrile conditions of obscure origin . . . It is probable that pyloric obstruction, spasmodic or otherwise, has no part in producing the condition, for in no instance has mechanical obstruction been demonstrated, and in four of the recorded cases the duodenum was dilated as well as the stomach". Morris<sup>3</sup> considers that the secretion of such enormous quantities of gastric fluid may be due to an error of nerve influence or to an alteration in the circulation in the organ. It cannot be said that our knowledge is much greater to-day. That the condition has followed operations involving the solar-plexus area and has occurred after operations on the kidney—perhaps from the pressure made to bring the organ into the wound—does not necessarily show its cause. With its great mortality it will turn the scale in cases of the wiring of abdominal aneurysm, and it seems to me that in such cases, and certainly when the sac is in the region of the cœliac axis, a gastro-enterostomy should be done at the time of the wiring. In most of such cases the anterior operation will necessarily be chosen.

The present condition of *Case 5* (female, aneurysm of ascending part of arch) is that the patient is living and shows pulsation in the chest and suffers pain. This is 5 years and 10 months after the first, and 4 years and 7 months after the second operation (Sir D'Arey Power).

It may be mentioned here that in 41 fatal cases of aneurysm of the abdominal aorta investigated consecutively and which had not been treated surgically, the mean age was 35.6 years (standard deviation 7.9), the mean 'clinical duration' 13.5 months (standard deviation 9.6), and that the longest noted was only 42 months. A large number of cases have been investigated since these figures were compiled, and I hope to be able to give them at a later date. Sir William Wheeler's two successful cases and Lawson's case are entirely beyond these figures, and so were the cases recorded by Langton (11 years 10 months) and Morse (4 years) wired by the older method. It is now clearly proved that the dull-gilt wisp is sufficient to initiate active clotting in a saccular aneurysm even in an actively syphilitic patient, and that it is unlikely to project through the opening of the sac. The wires do not become dissolved or broken, and appear to cause no trouble after many years. It would appear that the employment of electrolysis is not essential to start the clotting, and the experiments of Dawson Turner<sup>4</sup> amply confirm this. If electrolysis is employed, he recommends zinc as the electrode. Macdonagh<sup>5</sup> may be read in this connection. A simple way would be to coat alternate strands of the wisp with zinc.

## II. ANEURYSM OF THE ABDOMINAL AORTA.

By C. JENNINGS MARSHALL, LONDON.

Mrs. L. G., age 46, a widow, nulliparous, referred by Dr. F. G. Chandler, had undergone bilateral salpingectomy twenty years ago following a miscarriage. Her mother had died of phthisis. There had been pain in the left



side and in the middle of the back for nearly two years; the onset was after an attack of influenzal pneumonia. It had become much worse since attacks of 'dysentery' four and two months back; there was constant aching, with exacerbations, the patient was unable to lie upon the left side, and during the last two weeks had had practically no sleep, and was beginning to find the suffering utterly intolerable. She begged for something to be done, at whatever risk.

ON EXAMINATION.—There was a swelling in the epigastrium, slightly to the left of the middle line, about the size of a tangerine orange, with expansile pulsation and a pronounced thrill. The femoral pulses were present and not obviously delayed. There was tenderness over the lower dorsal vertebræ, but no angulation was evident. The physical signs of the swelling did not alter in the knee-elbow position. Radiography showed a rounded opacity in the epigastrium corresponding with the swelling; no vertebral erosion could be made out. Dullness and defective air entry were noted at the left base. The Wassermann reaction was negative.

The diagnosis was made of aneurysm of the abdominal aorta, at or below the origin of the cœliac axis. In view of the thrill, it was thought a comparatively saccular form might be present, and therefore that the case was favourable for some attempt to induce coagulation. Wiring was agreed upon.

OPERATION.—Under local anæsthesia a left paramedian epigastric laparotomy was performed. The gastrohepatic omentum was painted with novocain 1 per cent, and then divided for a few inches. The stomach was drawn down gently, and the aneurysm came into view above the pancreas, which was slightly pushed forwards. Over a prominent area a small quantity of novocain was injected, and then a purse-string suture of fine silk was inserted and left loose. The aneurysm was now punctured with an upwards and backwards thrust; the trocar appeared to be freely mobile in the sac, as if there were little, if any, clot present; on removal of the stilette blood spurted over the end of the operation table. The loaded cartridge was then engaged in the cannula and the blunt stilette pushed onwards. To one's consternation jamming occurred. The cartridge was removed, the opening in the cannula stopped with the finger, the wisp removed from the cartridge, and then fed into the cannula wrong end first. The blunt stilette was then able to push it inside the aneurysm; the cannula was removed and the purse-string tied. There was no leakage of blood, and the abdomen was closed.

The unfortunate contretemps was due to defective manufacture of the cannula and cartridge, so that after the electroplating they did not register exactly. In the small gap so caused at the breech end the ends of the wisp, tending always to expand, had impacted. As the wisp was finally introduced wrong end first, it was, of course, to be feared that expansion would be defective, the ends of the wires being expected to catch against the aneurysmal wall and in any thrombus already present. This was substantiated by a radiogram taken ten days after the operation (*Fig. 61*).

The patient slept poorly the night after operation; for a few days there was considerable flatulent pain, and then there was pronounced abatement of the symptoms. The aneurysm, though no smaller, seemed to be distinctly harder.

Three weeks after the wiring, however, the pain again became intolerable, worse, if anything, than before; the swelling was distinctly larger, and had extended downwards. The patient got no sleep, could eat little, and constantly complained of flatulent pain, though there was no intestinal distention.

A second wisp was introduced a month after the first, the same technique being used; the apparatus had been altered and no hitch occurred. There was again relief for three or four days, but then the pain returned and became very severe, both in the back and in the abdomen. The patient constantly urged that there was flatulence which should be relieved by puncturing the



FIG. 61.—Aneurysm treated by wiring.

bowel to let the gas out. Death took place twelve days after the second operation; during the last twelve hours there was actually great abdominal distention.

**POST-MORTEM EXAMINATION.** This was made by Professor A. Piney. The upper abdomen contained blood to a small amount—about 8 oz. There was a most striking acute dilatation of the stomach and duodenum down to the third stage of the latter, where there appeared to be compression between the aneurysmal sac and the superior mesenteric vessels. The sac (*Fig. 62*) was the size of a fist, having extended downwards and to the right, carrying forwards the pancreas and the duodenum at its lower part. The origin appeared to be immediately below the coeliac axis. The neck was comparatively narrow, about  $1\frac{1}{2}$  in. by  $\frac{3}{4}$  in. Slight leakage had taken place on the right

side, nearly 2 in. from the point at which the trocars had been introduced. The whole of the aorta showed severe patchy mesarteritis, and there was a second small aneurysm at the level of the left bronchus which had been compressed, with collapse and carnification of the lower lobe of the left lung.

The first wisp was seen to have reached to the neck of the sac and its 'handle' to be protruding slightly into the lumen of the aorta. It had expanded but little, the wires being partly caught among each other, partly held by the walls of the sac. The whole surface of the wisp, however, was practically covered by very adherent flakes of firm fibrin which were most difficult to peel from the wire. The second wisp, its end in contact with the first, had partly expanded and was embedded distally in recent thrombus which almost completely filled the aneurysm, and projected by a small tongue into the lumen of the aorta. The cut surface of this thrombus showed irregular whitish laminations, and its periphery was becoming adherent to the wall of the sac. In one place only could organized clot be detected, viz., as a thin layer anteriorly under the pancreas.

There was no aneurysmal affection of the posterior part of the aorta, no vertebral erosion. The viscera otherwise showed no striking abnormality. In Fig. 62 the wisps are shown displaced into the aorta. This occurred in sectioning the specimen. In the undisturbed state the expanded wisp lay entirely embedded in the whorled clot.

The *Spirochæta pallida* was demonstrated by the Levaditi method in sections of the aortic wall.

There seems to be little doubt that, as a result of the introduction of the wisps into the aneurysm, the sac had become filled by thrombus which was in early process of organization. Very careful investigation was made to see whether the puncture had led to rupture, and the question could be answered

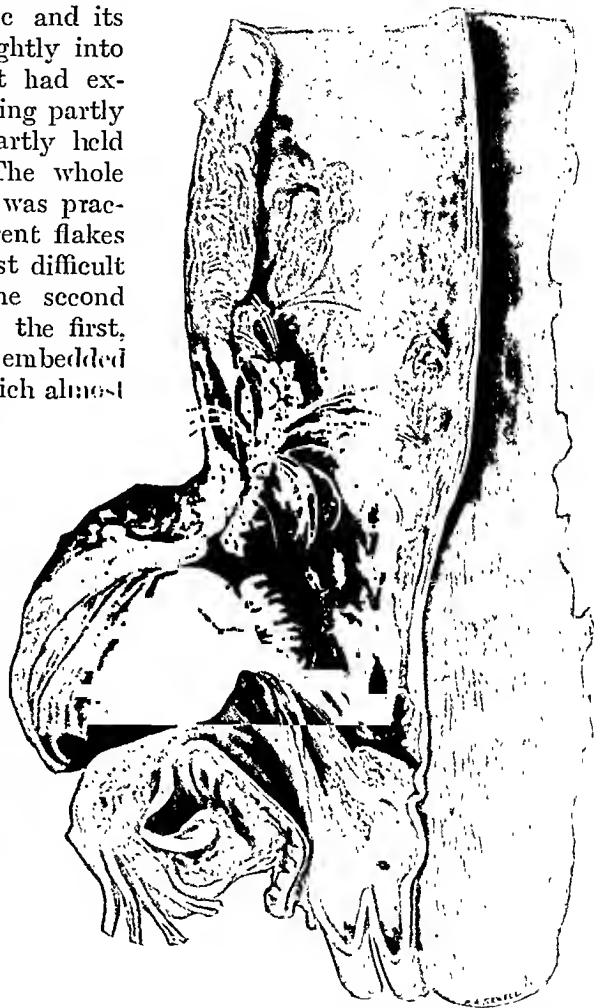


FIG. 62.—Post-mortem appearance of aneurysmal sac.

(Reproduced by permission from Marshall and Piney's "Textbook of Surgical Pathology": London, Arnold & Co.)

very definitely in the negative. The acute dilatation of the stomach may have been due, as is usually stated, to the pressure on the third stage of the duodenum; in this case there is, however, the possibility of severe disturbance of the splanchnic nerves by the sac. The persistent 'flatulent' pain seems strongly suggestive in this connection.

Despite the fatal outcome, one feels that the post-mortem findings fully justify the use of this method in abdominal aneurysm, particularly if saccular; and one would recommend its adoption in other cases.

### III. ANEURYSM OF THE ASCENDING AORTA.\*

By CECIL P. G. WAKELEY, London.

Wm. A., male, age 31, married, blacksmith and ex-sailor, admitted to King's College Hospital, Nov. 26, 1923; died Aug. 16, 1924.

About six months before admission, patient had a fall on the right shoulder, and shortly afterwards noticed a swelling in front of the chest, which gradually increased in size. The swelling has been associated with a continual aching pain in the chest, which was at its worst during the evening, and was just severe enough to keep him awake at night. Recently the pain had been more severe, and caused loss of appetite. The patient denied venereal disease, was a moderate smoker, and drank beer in moderation, but no spirits. Father and mother both alive and well, and he had two healthy children.

ON EXAMINATION.—The patient was a man of middle height and beyond average physique. There was a swelling on the upper anterior thoracic wall just to the right of the mid-line, about the size of an orange. The skin over the swelling was tense, red, and shiny. The swelling was visibly pulsatile, the pulsations corresponding in time to the apex beat. On palpation it was elastic, and the pulsation was felt together with the transmitted vibration of the closure of the heart valves; it was dull to percussion. On auscultation the heart sounds were heard somewhat distantly, and there was a soft systolic murmur, heard best over the lower pole of the swelling.

The patient did not suffer from shortness of breath. He had had a cough since leaving the navy eighteen months ago, which had got worse lately. He was not discharged from the service on account of aneurysm. The cough was dry, there was no phlegm, and at no time had the patient coughed up blood. There was diminished air entry in the right lung, most marked in the scapular region posteriorly. There had never been any difficulty in swallowing, and he had never noticed any change in his voice. The cords were normal. The Wassermann test was positive. Pulse 120, respirations 24, temperature 97°.

The diagnosis of aneurysm of the first part of the aorta was made; the sac had perforated the chest wall and was subcutaneous.

OPERATIONS.—On Nov. 30, 1923, under C. E., the lower limit of the swelling was exposed by a curved incision following the fifth rib, and a flap dissected up. A No. 2 wisp of wire from a Colt's apparatus was introduced into the

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\* This case has also been recorded in the *Proceedings of the Royal Society of Medicine*, 1923-24, Clinical Section, p. 18.

aneurysm from below. The character of the pulsations changed from expansile to thrusting. The after-progress was uneventful, the only feature about the swelling being the change in character of the pulsation. Cough and pain persisted. The wire was demonstrated in the sac by X rays.

On Dec. 31, 1923, the operation was repeated, another wisp of wire (No. 2) being introduced; the anæsthetic was rectal ether.

On Jan. 12, 1924, the swelling appeared to be somewhat smaller than on admission. The patient was free from pain, and the cough was much better. The blood-pressure was 95 systolic and 65 diastolic.

On Jan. 25 the patient got up and felt no ill effects on slight exertion. At this time a systolic murmur replaced the first sound at the apex, and a quiet murmur, systolic in time, was audible over the upper part of the tumour. Electrocardiogram was normal. Medical treatment consisted in administration of potassium iodide and mercury. The patient was discharged.

The patient was re-admitted on May 21. There was now a second swelling slightly above and to the left of the original one, and smaller. The original swelling had not increased in size since his discharge. The second swelling was similar to the first, and on auscultation over it a blowing systolic murmur was audible. Pulse regular, 110, respirations 28, temperature  $97.6^{\circ}$ .

On May 23 under gas and oxygen anæsthesia two No. 2 wisps of wire were inserted, one into either swelling. For a short time afterwards the pulse improved in volume, and the swelling on the left decreased in size and became less expansile. During this time the temperature rose in the evenings, and a small collection of pus formed under the skin. This cleared up satisfactorily, and the patient was discharged on June 28. He was re-admitted on Aug. 16 in a weak condition, with a dry, intractable cough. At the site of operation there was an unhealed wound from which blood leaked continuously. The bleeding rapidly became more profuse, and the patient died on Aug. 16, eight and a half months after the first operation.

**POST-MORTEM EXAMINATION.**—The pericardium was obliterated by dense adhesions, which were universal. There was no thickening of the parietal pericardium. The mitral valve admitted three fingers. The aortic valve was normal and competent.

There was a large aneurysmal sac on the ascending aorta. The cavity was about 4 in. in diameter, and the mouth, which was situated about 1 in. above the aortic valve, was about  $1\frac{1}{2}$  in. in diameter. This cavity passed forwards to the front part of the sternum, and was filled with post-mortem—'chicken-fat'—clot. The walls of the sac were covered with ante-mortem laminated clot. The aorta was dilated, and formed a cavity which commenced 1 in. above the aortic valve and extended in a backward direction towards the root of the left lung for a distance of about 3 in. This cavity also contained post-mortem clot. There was an abscess cavity, which did not communicate with the aorta, opening on the chest wall at the site of the aneurysm.

There was syphilitic inflammation of the root of the aorta, but only a slight degree of atheroma was present. The coronary arteries were patent. The aneurysmal sac contained the wisps introduced at the operations and

laminated clot had formed around them. All the wisps had expanded properly, and no part of any of the wires was uncovered.

The pleuræ showed some recent adhesions and contained a little fluid. The apex of the right lung was adherent to the chest wall, and the upper lobe had collapsed owing to the pressure of the aneurysm. The right middle lobe was adherent to the mediastinum in contact with the sac. There was a small scar on the right kidney.

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## EXTRAVASATION OF BILE.

BY ZACHARY COPE, LONDON.

*(Being a Hunterian Lecture delivered before the Royal College of Surgeons of England on February 2, 1925.)*

THE body is provided with various kinds of tubes which convey the different fluids to their appointed destination. As the result of injury or disease any of these tubes may leak, so that extravasation of a fluid is a common pathological phenomenon; in the case of blood it is an every-day occurrence, with urine not an uncommon happening, whilst it is only too frequent for the surgeon to have to deal with extravasation of gastric or duodenal contents. Extravasation of bile is less common, but not very rare; yet it is most difficult to find anything more than a passing reference to the subject in any but the Continental literature. English and American surgeons must be well acquainted with the condition, but have, so far as I can find, not thought it worthy of their detailed consideration. This statement applies with particular force to that variety of extravasation in which no perforation of the biliary tract is discovered at autopsy or operation. When a series of such cases came under my observation, therefore, a further consideration of the subject seemed necessary.

The anatomical course of the bile-duct is well known. After leaving the liver by the right and left hepatic ducts, the bile flows through the common hepatic duct which is formed by their junction; the common hepatic is joined by the cystic duct, and the combined trunk—the common bile-duct—courses down in the right free edge of the gastro-hepatic omentum, behind the first part of the duodenum, and behind or through the head of the pancreas, to join the second part of the duodenum. The gall-bladder may be regarded as a blind dilated recess of the cystic duct.

The peritoneal relations of the ducts are important. The gall-bladder and cystic duct are covered by peritoneum over the greater part of their surface, but there is a varying bare area on the side towards the liver and portal fissure. The hepatic ducts and upper third of the common bile-duct are clothed with peritoneum, but the lower two-thirds of the choledochus are retroperitoneal—a fact which we shall see has a rather important bearing on some cases. The lower end of the common duct is, in a considerable percentage of cases, embedded in the substance of the pancreas, but often it merely lies in a groove on its posterior aspect.

The structure of the gall-bladder and bile-ducts consists of a mucous membrane, a fibromuscular wall, and a peritoneal coat (*Fig. 63*). The muscular coat is not very thick, and, so far as my observation goes, seldom undergoes hypertrophy in order to overcome obstruction. In this it differs greatly from the musculature of the intestines and the ureters. Obstruction of the common bile-duct is generally accompanied by a dilatation and thinning of the wall of the duct above the obstruction.

The mucosa of the biliary tract is furnished with a large number of mucous glands. These glands are frequently large, and send their ramifications down to and—as Luschka<sup>1</sup> showed sixty years ago—sometimes *through* the muscular coat. I am told that the occurrence of these glandular acini under the serosa of the gall-bladder is well known to pathologists and is sometimes used to puzzle the student, who may naturally think that carcinoma is beginning; and yet I cannot find this anatomical point referred to in any English pathological or anatomical text-book, though it may be of some considerable pathological importance. (*See Fig. 63.*)

It is well to remember that the first and second parts of the duodenum contain chiefly bile, except for a short time after meals. The duodenum, liver, colon, and gall-bladder are in close relationship, and adhesions readily form which tend to limit extravasation.

Bile is secreted at low pressure, and though, if there be a lateral opening in one of the duets, bile will escape, yet so long as there is no obstruction beyond the opening, the fluid tends to run in the correct channel and the unnatural opening tends to close. The surgery of the common bile-duct would otherwise be an unsatisfactory procedure, for it is a common operation to open the duct and leave it unsutured or only partly sutured. So long as a drain is put down to the duct it heals readily. No surprise should therefore be felt if an injury of the bile-duets or gall-bladder should heal up fairly quickly and leave little trace of its exact position. Since this point has bearing on our subject, I will illustrate it by recounting a case which was under the care of my colleague, Professor Pannett, two years ago, and which he kindly gave me permission to mention.

*Case 1.*—John S., age 10, was knocked down by a motor-car which doubled him up and 'winded' him. He vomited a little blood. He was kept in bed for two days, then got about a little, but about twelve days after the accident there was a recurrence of abdominal pain and the abdomen became distended. When admitted to St. Mary's Hospital two weeks after the accident, his abdomen was distended, with general rigidity and tenderness. At operation, Professor Pannett found half a bucketful of fluid in the peritoneal cavity. The fluid contained much bile. There was no rupture of the liver, but an *undetermined* lesion of the bile-duct in the region of the junction of the hepatic, cystic, and common bile-duets. There was soft inflammatory infiltration in this region, and the exact site of the escape of bile was not detected. The gall-bladder was collapsed. The gall-bladder and the site of injury were drained, and a good recovery followed.

There are many similar instances published which show the natural tendency to closure of any rupture of the duets. A certain amount of bile escapes, but a deposit of fibrin occurs and seals the opening. There is no difficulty in appreciating how, in many cases, extensive extravasation is prevented by three factors: (1) Low pressure of bile secretion and possible intermittency of the secretion; (2) Natural tendency to closure of the opening; (3) The readiness with which adhesions form to neighbouring viscera. This tendency to closure is not so well marked in the other muscular tubes.

Bile may be extravasated from any of its containing channels, and will pursue a different course according to the situation of the part from which it escapes. Since by far the greater surface of these channels is covered by peritonium, it is to be expected that the majority of cases will show leakage



into the peritoneal cavity, and such is the fact. Yet there are a certain number of cases in which the bile finds its way chiefly into the retroperitoneal tissues. This may occur primarily from a part of the biliary tract which is uncovered by peritoneum, or secondarily by an intraperitoneal collection bursting through into the tissues behind the peritoneum. Amongst the total of 384 biliary fistulæ enumerated by Naunyn,<sup>2</sup> there were but 4 which were retroperitoneal. I do not doubt that others have seen such cases, but I



FIG. 63.—Section through the wall of a gall-bladder, clearly showing a mucous gland outside the muscularis mucosæ. (Kindly lent by Professor Newcomb.) ( $\times 37$ .)

have not been able to find any clinical account of such a condition in modern surgical literature. My two cases were as follows:—

*Case 2.*—Mrs. K. P., age 32, was taken with acute epigastric and right iliac pain at 8 a.m. on June 9, 1922. The epigastric pain ceased next day, but the iliac pain continued, and there was slight pain on micturition, and also over the front of the right clavicle on breathing.

When I examined her at St. Mary's Hospital on June 10 there were tenderness and an indefinite swelling in the right loin, where the muscles were rigid. There was not so much tenderness in the right iliac fossa. There was pin-stroke hyperæsthesia above the outer part of the right Poupart's ligament and the anterior part of the right iliac crest. At 10 p.m. on the 10th I operated by a right paramedian incision. The appendix was normal, but free bile was discovered in the peritoneal cavity. There was fibrinous lymph under the liver, and some lymph in front of the liver (accounting for the clavicular pain), and an infiltration with bile of the posterior parietal

peritoneum, which was of maximal intensity just to the right of the descending duodenum in front of the renal pelvis. There were no gall-stones, and I found no perforation of the gall-bladder or bile-duets. As I wiped the posterior peritoneum dry it almost immediately wept bile once more, though I could see no opening in the peritoneum.

I drained the gall-bladder and the subhepatic pouch, and an uninterrupted recovery took place.

The noteworthy points in this case are the absence of jaundice, the possibility of localizing the lesion by the anatomical symptoms, and the failure



FIG. 64.—Section through the lower end of the common bile-duct, showing the absence of the muscularis mucosæ in one place and demonstrating the deep penetration of the mucous glands. (Section kindly cut for me by Professor Newcomb.) ( $\times 37$ .)

to find a perforation which presumably must have existed behind the peritoneum. Such a favourable termination was lacking in the second case, which was nevertheless in many ways more remarkable.

*Case 3.*—Mrs. A., age 50, was admitted to the Bolingbroke Hospital on May 27, 1921, with a history that forty-eight hours previously she had been taken with abdominal pain—first epigastric, then right iliac. She vomited. When I saw her soon after admission I noted that she was a very stout woman, and that there was a fomentation scald in the right iliac region, where there were tenderness and muscular rigidity. Pin-stroke hyperæsthesia was noted as a band about 2 in. broad above Poupart's ligament and the iliac crest on the right side as far back as the outer margin of the erector spinæ muscle. There was pain on extension of the thigh.

Appendicitis was diagnosed, and the abdomen opened by a right-iliac-oblique incision. No sign of recent appendix inflammation was noted, however, but the tip of the organ was discovered separate from the rest—clearly from old perforative inflammation.

The most noteworthy observation was œdema of the posterior parietal peritoneum and retroperitoneal tissues. On incising the posterior peritoneum we found extensive extravasation of bile into the retroperitoneal tissues round the right kidney, duodenum, and down to the right iliac fossa; the bile was green, and glittered with innumerable cholesterol crystals. The incision was then prolonged upwards, and a stone found in and removed from the gall-bladder. No perforation was found, but there were omental adhesions, and it was difficult to explore thoroughly. There was no bile in the peritoneal cavity. Drainage down to the gall-bladder and retroperitoneal tissues was carried out.

The patient died ten days later, but no autopsy was obtained.

These two cases well illustrate mild and severe forms of retroperitoneal extravasation, and for their causation one must assume a leakage from the retroperitoneal surface of the gall-bladder or common bile-duct. Such leakage causes irritation of the pelvis of the kidney and the upper end of the psoas, whilst there is likely to be hyperæsthesia of the twelfth dorsal segmental area.

Intraperitoneal extravasation of bile is more common, and can be caused by injury or disease of the gall-bladder, bile-ducts, or duodenum. There is no doubt as to the immediate cause of the extravasation in some cases. Penetrating or subcutaneous injury of the biliary tract leaves little room for doubt, for, even though at operation the actual site of rupture may be difficult to detect, the connection with the injury is usually too consecutive to be other than consequential. Perforation of the gall-bladder due to gangrene or ulceration may also lead to extravasation, but the leakage is generally localized to the right hypochondrium by limiting adhesions.

Sometimes the fluid which escapes from the duodenum on perforation of an ulcer appears to be almost pure bile, and may cause a suspicion of rupture of the biliary tract. Pure bile appears to cause less peritoneal irritation than the ordinary fluid which escapes after perforation of a duodenal ulcer, and the symptoms may be less brusque in onset. The afore-mentioned causes are clear, and there is little need to furnish illustrative cases, though such could easily be afforded. There remains, however, a considerable group of cases in which there is a plentiful biliary extravasation, but neither at operation nor at autopsy (if such takes place) can any perforation of the biliary tract be discovered. This condition has aroused discussion, chiefly in Germany, where exhaustive and exhausting treatises have been written, with rather inconclusive results. During the past few years I have had six such cases under my care. To these I will add another patient upon whom my colleague, Mr. Bryan, operated.

### SYNOPSIS OF CASES.

*Case 4.*—M. H., age 55, female, was seen by a colleague on Aug. 23, 1920, suffering from agonizing abdominal pain and vomiting.

**CONDITION.**—Aug. 23: Pale, shivering, and in great pain. Legs drawn up. Respiration 30, pulse 100, temperature 102°. Slight jaundice. Slight epigastric rigidity. On Aug. 24 there was distention, which was increased next day, when the pulse was 118, respiration 30, and pain was less. I was then asked to see her and decided to operate.

**OPERATION.**—Subumbilical incision: inflamed intestines found. Epigastric incision: free bile in peritoneum. Gall-bladder adherent to duodenum. No stone found. Gall-bladder aspirated and drained. Drainage also of pelvis and gall-bladder recess.

**RESULT.**—Recovery.

*Case 5.*—Female, age 60. In September, 1921, the patient had abdominal pain, first generalized, then localized to right hypochondrium.

**DIAGNOSIS.**—Acute cholecystitis.

**OPERATION.**—Abdomen full of green fluid. Lymph round first part of duodenum, but no perforation found. Drainage suprapubic and gall-bladder pouch.

**RESULT.**—Died.

*Case 6 (Fig. 65).*—C. M., age 55, male. A history of jaundice for three weeks; on the morning of June 28, 1922, there was an acute onset of abdominal pain and distention.



FIG. 65.—Biliary tract in *Case 6*. Common duct larger than gall-bladder; it contained 40 stones, some as large as a hazel nut. No perforation found. ( $\times \frac{2}{3}$ .)

**CONDITION.**—June 28: Distended; much free fluid in abdomen; tender right lumbar region; very large liver; cardiac murmurs. On the next day there were hæmatemesis and hæcough.

**RESULT.**—Died. (No operation performed.)

**POST-MORTEM.**—General peritonitis. Bilious purulent exudate; no perforation found; common duct enormously dilated, with many stones; healed tubercle both lungs; aortitis and small sacular aneurysm; hydatid cyst of right kidney; adherent pericardium; patent foramen ovale; tape-worm.

*Case 7.*—Female, age 79, was seen on Aug. 20, 1923, with a history of six days' abdominal pain; acute onset, then remission, and exacerbation.

**CONDITION.**—Signs of general peritonitis. Distended abdomen; congestion of lungs; weak heart action. (Consultation with a physician.)

**OPERATION.**—Free bile in peritoneal cavity, especially in right hypochondrium. Lymph over ascending colon; no perforation found; drainage of peritoneal cavity only.

**RESULT.**—Died same day. (No autopsy.)

*Case 8.*—Female, of middle age, seen on Nov. 19, 1923, had a history of indigestion for years, sometimes with jaundice; attack of abdominal pain on Nov. 10. There was a remission of symptoms, but she became worse again on the 17th, with frequent vomiting, and pain on top of shoulder.

**CONDITION.**—Nov. 19: Abdominal facies; looked moribund. Pulse 108, almost imperceptible. Abdomen tumid and tender all over.

**DIAGNOSIS.**—Ruptured gall-bladder or duodenum.

**OPERATION.**—Right paramedian incision: bile in peritoneal cavity; no perforation of duodenum; dense mass of adhesions in biliary recess. No gall-stones felt, but gall-bladder impossible to identify. Drainage of biliary recess and of pelvis.

**RESULT.**—Against expectation the patient improved, and left the nursing home in a month. Soon after had acute attack of abdominal pain, and succumbed.

*Case 9.*—S. H., female, age 55. When seen on April 5, 1924, had had five weeks' abdominal pain, with vomiting, sweating, and attacks of faintness for one week.

**CONDITION.**—Subnormal temperature; distended abdomen; pulse normal.

**DIAGNOSIS.**—Intestinal obstruction.

**OPERATION.**—On opening abdomen, bile welled up; large liver and gall-bladder. Three-quarters of a pint of bile aspirated from gall-bladder; two gall-stones removed from gall-bladder. No perforation of duodenum or bile passage discovered. Gall-bladder and peritoneal cavity drained.

**RESULT.**—Died within twenty-four hours. (No autopsy.)

*Case 10 (Mr. Bryan's case).*—Male, age 26. History of flatulence for some weeks. At 5 a.m. on June 14, 1913, patient had acute right hypochondriac pain going through to lumbar region; vomited. There was a history of a similar attack six months before; never jaundiced.

**CONDITION.**—Pulse 104, temperature 101°. Slightly rigid and tender right hypochondrium; chest nil; rectal examination negative; no jaundice. June 15: slight jaundice. Collapsed in afternoon and became distended.

**OPERATION.**—Acutely distended pink gall-bladder. Golden bile staining of portal fissure region before gall-bladder opened. No perforation found; no stone found. Gall-bladder aspirated and drained. No peritonitis.

**RESULT.**—Recovered.

In none of these cases was any definite perforation found. It will thus be seen that the clinical symptoms of intraperitoneal extravasation of bile are very variable. The picture is that of an acute or subacute peritonitis originating in the right upper quadrant of the abdomen. The onset may be acute or gradual; jaundice may be present or absent; gall-stones may be found in the gall-bladder and ducts, or the biliary passages may contain nothing but bile; there may be a subnormal, normal, or elevated temperature; and abdominal tenderness, rigidity, and distention vary greatly in degree. In the midst of this variability there is one feature which is almost constant, i.e., the remission of symptoms after the first acute onset. Remission of symptoms is unfortunately a common occurrence in many acute abdominal disorders, but perhaps in none is it so marked and dangerous as in extravasation of bile. Omental adhesions form, and for the time easily stop the progress of the bile, which is under low pressure; the patient and doctor think more lightly of the condition, undue liberty and exercise are allowed, and extension of the disease occurs. Only thus can we account for the advanced stage of the disease in several of the cases under review. Clearly the remedy is to consider every case of right hypochondriac peritonitis as dangerous.

## CAUSES OF BILE LEAKAGE.

We will now consider the view held in different quarters as to the reason for the leakage of bile in these obscure cases.

The first discussion and experimental work on the question was published in 1911 by Clairmont and Haberer.<sup>3</sup> Their interest was aroused by finding free bile in the peritoneal cavity of a man who had stones in the cystic and common duct, but in whom no perforation was found in the biliary tract either at operation or at autopsy. Their experiments seemed to show that ligation of the common duct in dogs would cause exudation of bile into the peritoneal cavity without perforation of the biliary tract. They explained the leakage as a filtration through the walls of the gall-bladder or ducts. They assumed "a pathological process of the biliary passages not microscopically demonstrable" which permitted the filtration.

Following the researches a host of eager observers and disputants entered the field. The filtration theory was supported by Schievelbein,<sup>4</sup> who thought the channels of Luschka's glands a likely site for leakage; but opposition came from many who thought it more likely that a minute perforation could be overlooked. The suggestion was put forward by Wolff<sup>5</sup> that there might be leakage from the intrahepatic canals on the under surface of the liver, whilst Johansson<sup>6</sup> theorized as to a possible hypertrophy of the lymphatic canals.

The filtration theory was criticized in 1921 by Ritter,<sup>7</sup> who remarked that "the objection that though none be found there can yet be some perforation must be reckoned with"; and again, "only very careful macroscopic and microscopic examination can give proof as to whether perforation existed or not".

Burchardt<sup>8</sup> in 1923, in an elaborate article, came to the conclusion that "the occurrence of an authentic biliary extravasation or biliary peritonitis without perforation is not yet proved"; and that "a more plausible view than the filtration theory is the suggestion that the perforation opening was not found or was already healed". My own observations lead me to agree with this suggestion. It would be altogether contrary to our knowledge of involuntary muscular tubes to allow that they would permit filtration of their contents even when seriously damaged, so long as there was no perforation. Much more likely is it that there is a minute perforation which allows escape of bile when under tension. After the escape of sufficient bile to relieve tension the opening may be sealed off by lymph, so that no more fluid enters the peritoneal cavity. This process may occur from time to time until the symptoms are so serious that surgical interference becomes imperative. The minute perforation is sometimes in the gall-bladder, but undoubtedly sometimes in the ducts, since in several cases the gall-bladder was tensely distended but not leaking. The nature of the pathological process which causes the minute perforation must be presumed to be a small area of infective or traumatic ulceration of the mucosa. The biliary tract is very prone to infection, and is liable to trauma of a special type, i.e., that caused by a passing stone. It is needless, and indeed impossible, to invoke a gall-stone

as the only cause of injury to the duets, for in many cases no stones are found and no definite history of colic is given; but that it is a possible and even likely cause in some cases I have no doubt. On one occasion when I opened a man who showed signs of right hypochondriac peritonitis, and who gave a perfectly typical history of biliary colic with sweating and writhing for several hours, I found no perforation of the duodenum or gall-bladder, but much lymph round those viscera, and a much thickened right free edge of the gastrohepatic omentum, with some green bile showing under the peritoneal covering of that structure immediately over the position of the common duct. Though no gall-stone was found or obtained from the fæces, I believe that a stone had passed, had stretched the common duct, and allowed slight leakage of bile. It is a tempting hypothesis to assume that the glands of Luschka provide a ready channel for spread of bacterial infection and for leakage of bile. (*See Figs. 63 and 64.*)

There is no clinical doubt that the peritoneum is the strongest safeguard against intraperitoneal extravasation. If there is ulceration within the bile-duets, no leakage is likely to occur into the peritoneal cavity until the peritoneum itself is damaged. It is not uncommon to see a very tensely distended gall-bladder with severe ulceration of the mucosa, but a fairly healthy silvery-white peritoneal covering which allows no leakage.

**The Differential Diagnosis** has to be made from the other causes of spreading peritonitis originating in the right side of the abdomen. The chief conditions are: cholecystitis, perforation of a duodenal or pyloric ulcer, appendicitis, acute or subacute pancreatitis.

In the late stages, when there is an extensive peritonitis, one cannot exclude intestinal obstruction except by considering very carefully the previous history. In cases of appendicitis and perforated ulcer the history is usually sufficient to guide to a correct diagnosis. I see no way of differentiating many cases of cholecystitis and pancreatitis from the results of a leakage of bile.

So great is the difficulty in diagnosis that Ritter in 1921 stated that the correct diagnosis of biliary peritonitis had never been made before operation. That statement needs some modification, for it was in August, 1920, that I diagnosed one of my cases as peritonitis from a ruptured gall-bladder and found biliary peritonitis at operation. On referring to the symptoms in this case it will be found there were slight jaundice and a history of epigastric pain and increasing symptoms of peritonitis to guide me. In my other cases I made incorrect diagnoses. I think in the future diagnosis may possibly be helped by finding bile in the blood.

**Treatment.**—The treatment of biliary extravasation is by operative measures. The bile must be allowed to escape from the peritoneal cavity so far as is possible, and any tension in the biliary passages must be reduced. In a patient who is very ill, merely drainage of the peritoneum and of the gall-bladder is called for.

If the biliary duets appear normal and the bile appears to be escaping from the gall-bladder, it is a natural procedure to remove the gall-bladder,

provided the patient's condition warrants it. Any stones in the common duct should be removed.

With the present late-coming of these cases, however, the mortality is very great. Six out of ten of the cases mentioned in this paper succumbed. It is quite clear that the curing of acute abdominal disease depends chiefly upon early diagnosis.

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## CARCINOMA OF THE VERMIFORM APPENDIX.

BY ERNEST H. SHAW, LONDON.

AN interesting primary tumour of the vermiform appendix is met with occasionally by the pathologist. It occurs usually as a small round yellowish growth situated, in nearly all cases, at the distal end of the organ. Microscopically the tumour is found to consist of colonies of rounded cells lying in well-marked alveoli, and often seen invading the muscular wall. In some specimens the cells actually reach the peritoneum. In addition to the round cells there are also present columnar cells which are arranged in a ring-like manner. These cells vary in number in different specimens, usually scanty, but they may be quite numerous. Vaeuolation of the cell bodies is common.

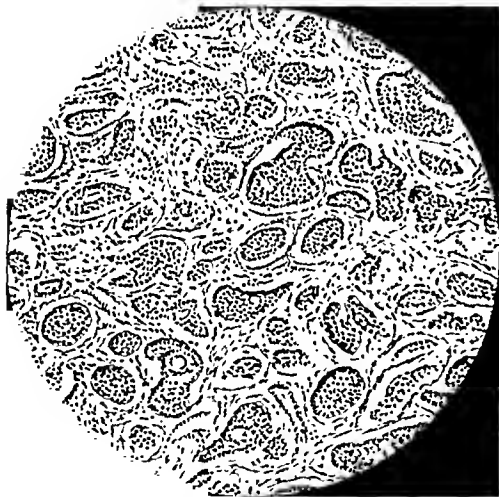


FIG. 66.—Case 1. Colonies of cells in alveoli. Note ring of nuclei around many of the masses suggesting immature columnar cells, and vaeuolation in the midst of some groups. Matrix composed of fibrous tissue. Low-power view. ( $\frac{1}{3}$  obj., No. 4 eyepiece.)

Like other observers I have been at a loss to account for the origin of these cells. What particular kind of cell in the wall of the appendix could one look upon as being the parent cell?

In writing my reports on these tumours, I have been obliged to state that the cells of the growth have the general arrangement and infiltrating character of carcinoma, and to modify this by adding that the clinical course of the growth is usually that of an innocent tumour—clearly an unsatisfactory state of affairs both for the pathologist and clinician.

The problem as to the true nature of the tumour has led to much investigation and speculation

by pathologists, the points of interest and importance being: the origin and type of cell giving rise to the tumour; and, Is it innocent or malignant in its clinical behaviour?

Some pathologists have labelled this variety of tumour by that much-abused term endothelioma, a name under which so many tumours are classed when the pathologist is in doubt whether he is dealing with a carcinoma or sarcoma. I have always been at pains to avoid using this term, and sought every avenue of escape from it. In spite of this dislike of the term, I have fallen to the temptation and used it occasionally in a fit of desperation and

as an easy way out of a difficulty. There can be no doubt that endothelial cells do proliferate like all other cells of the body and give rise to tumours, but I could never see any real justification for labelling these primary tumours of the appendix with the term endothelioma. Endothelial cells are oval or spindle in shape. The appendix-tumour cells are round and columnar.

After my last specimen was received and reported on in the usual way, I began to ponder over these growths again, and to wonder whether I could discover anything definite as to their real origin and nature. The important point of after-history of the patients from whom the tumours were removed seemed a valuable one to investigate. I went through my private records and those in the Pathological Department at the Royal Northern Hospital: the list of cases, with all available and relevant details,



FIG. 67.—Case 1. Edge of same growth, showing small round-celled infiltration and several Lieberkuhn's glands in the upper part of the field. The mucous membrane is invaded and partly destroyed at this spot. (Same magnification.)

is given in the following pages.

The outstanding feature revealed in the clinical data is the presence in almost every case of symptoms pointing to some trouble in the region of the appendix. In some cases there is evidence in the specimens of co-incident inflammatory changes, while in others the appendix was normal apart from the presence of a tumour. In regard to the latter group it is perhaps permissible to advance the theory of interrupted or irregular peristalsis of the appendix due to the presence of a tumour as a cause of the symptoms.



FIG. 68.—Case 1. Several large groups of cells showing vacuolation and an attempt at tubular formation. The cell masses have shrunk away from the alveolar walls during the process of preparation of the section. High power. ( $\frac{1}{4}$  obj., No. 4 eyepiece.)

All the specimens exhibited some kind of swelling or tumour. In several it was not possible to tell by naked-eye inspection whether the swelling was due to simple fibrous hypertrophy with obliteration of the lumen—a common condition accompanying chronic appendicitis—or to a new growth. In others the yellowish colour of the tumour prompted the inquiry as to the possibility of tuberculous disease being present. The only specimen in my series in which a tumour was present in the body of the appendix was one apparently composed of two nodules (*see Case 9*). They were lying side by side, and had ulcerated on the inner surface.

In an effort to elucidate the nature and origin of the tumour cells, I studied closely all the microscopic sections in my possession, and found there was a striking similarity in the type of cell present in all the speci-

mens, and also in the arrangement of the cell masses. As stated above, the cells are mostly round in shape, and some of them are rather large; they lie in well-defined alveoli. The nucleus is small and round. The number of cells in the alveoli varies from half a dozen or so to a hundred or more, and they are usually packed closely together. The alveolar walls are composed of well-marked fibrous tissue in the main tumour, but where the cells have invaded the wall of the appendix, the muscular tissue of that structure forms the boundaries of the alveoli.

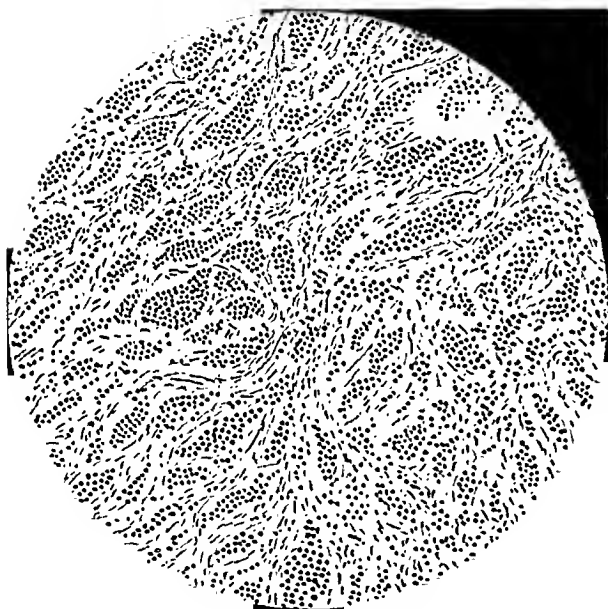


FIG. 69.—*Case 3*. Illustrating the general alveolar distribution of the tumour cells, which in this field are all about the same size and round in shape. Low power. ( $\frac{2}{3}$  obj., No. 4 eyepiece.)

These tumour-cells are quite unlike those lining the mucous membrane, which are tall and columnar in shape. What, then, is the parent cell? The only large round cells met with in the normal appendix are those composing the sympathetic ganglia and the island cells in the lymph nodules. The question arises: Are either of the latter the parent cells of the new growth? Against this theory of origin is the fact that these two varieties of cell belong to the connective-tissue group, and cellular tumours of connective-tissue origin are composed of cells glued together, as it were, by an interstitial substance, and not distributed as groups or colonies lying loosely in alveoli such as occurs in tumours derived from epithelium.

The alveolar distribution of the cells in the appendix tumours is so striking that I felt convinced that the cells must be of epithelial origin. The

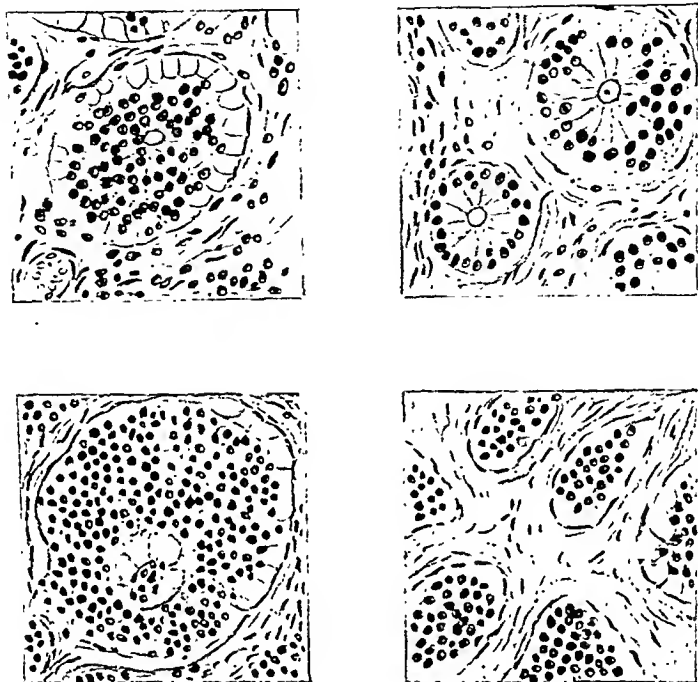


FIG. 70.—Case 3. Four small fields selected to show the variation in type of cells. In (A) and (C) vacuolation of the cells is well marked, and there is a tiny central lumen in (A). (B) Shows two well-formed tubules in cross-section. (D) mainly composed of rounded cells closely packed. High power. ( $\frac{1}{6}$  obj., No. 4 eyepiece.)

presence of columnar cells in parts of the tumours strengthened this opinion. I was also struck by the close resemblance of the microscopic pictures to those

FIG. 71.—Case 3. Main growth well defined within the muscular wall. Below on the right it has penetrated the wall and forms a secondary smaller mass. Outlying deposits of tumour cells are indicated in the muscle and peritoneum by tiny black dots. ( $\times 4$ )



of many breast carcinomata. In most of the latter the predominant cell is round (spheroidal), but a varying number of cubical and columnar cells are

generally seen in some parts of the sections. Following this line of thought while examining the sections of the appendix tumours, I began to appreciate more fully the significance of the columnar cells, which were seen in varying number. A group of columnar cells arranged in a ring, with the clear protoplasm of each cell on the inner side and a small lumen in the centre of the ring, demonstrated well an attempt at tubular gland formation. These rings of cells may lie in the midst of the round cells which form the great bulk of the tumour, or be present in small alveoli of their own. Quite a number of such rings were found in one specimen. In another slide two rows of columnar cells with a space between the columns clearly indicated a tubular gland cut in longitudinal section (*see Fig. 76*).

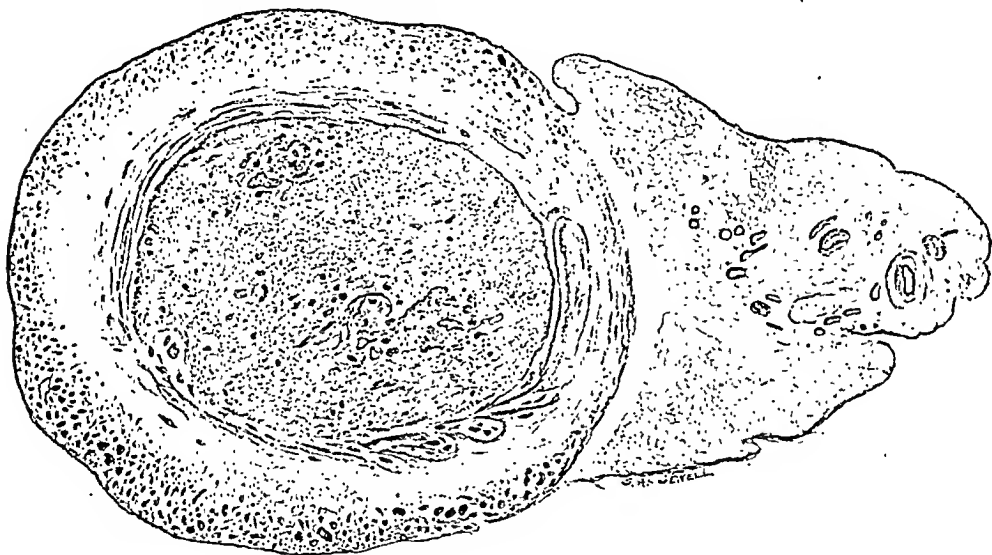


FIG. 72.—Case 5. Similar location of main tumour within the appendix; it is unusually fibrous and contains numerous blood-vessels. Offshoots of tumour cells are seen invading the muscular wall, and a wide area of small colonies is seen in the outer layers of muscle and in the peritoneum. The middle portion of the muscle is practically free from growth. (about  $\times 12$ .)

Before accepting the theory of epithelial origin of the appendix tumours it will be necessary to explain the presence of the round cells in such predominant numbers. Can it be proved that these cells are epithelial in origin? Do they arise from the same type of cell as do the columnar cells? If so, why are they round and not columnar in shape? I think a satisfactory answer to this question is to be obtained by comparison with the composition of carcinoma of the breast and other parts of the body. As referred to above, most cases of carcinoma of the breast show columnar cells in addition to the predominant spheroidal cell. The same mixture of cells occurs in some specimens of nasal and antral carcinomata. It is also seen in the stomach. It appears therefore that while some cells attain their typical columnar form in the appendix tumour, the main bulk of them have not advanced in development beyond their primitive circular shape.

If we accept this theory of origin from columnar epithelium, how can we explain the striking difference between the ordinary intestinal growths with their multitudes of typical columnar-celled glands and the tumours of the appendix under review?

I venture the opinion that, whereas the former growths undoubtedly arise from the surface layer of tall columnar cells, the appendix tumours originate from deep-seated epithelial cells which have been cut off in the early stage of foetal life. This is, of course, in conformity with Cohnheim's theory of 'latent embryonic rudiments' being a source of origin for certain tumours. I feel sure that this explanation must be the correct one.

It may be asked why, if a few columnar cells are separated in the course of development, they do not grow into well-marked columnar cells in a subsequent tumour? The answer to this question is simple. In very early foetal life all the cells are round in shape; this is the basic type, and from it spring all the higher forms during the development of the compound organism. Failure to form typical columnar cells from a rudimentary deposit of parent cells shows a weakness in developmental power. The cells may multiply, but, with the exception of a few which reach the columnar stage, they seem incapable of attaining the higher type. This weakness in the power of full development of cells is seen in most specimens of carcinoma—a typical columnar-celled growth usually contains many round epithelial cells.

From the evidence given above, I feel justified in asserting that this particular variety of primary tumour of the appendix belongs to the epithelial group.

Another interesting feature of these tumours is the variability in the number and distribution of small round inflammatory cells. In many fields under the microscope there is an entire absence of these little cells, and in others a few are seen. In several specimens they are numerous.

A feasible explanation for this variation is that the tumour is at first deep-seated and well away from the lumen of the appendix, thus well shut off from septic infection. As it enlarges, the mucous membrane is pushed away and stretched over it. The thinned membrane is in this way damaged, and infection by micro-organisms from the gut takes place. Small round-celled invasion then occurs in the usual manner. A coincident attack of appendicitis is another cause.

The next problem that confronts the pathologist is: Is the tumour innocent or malignant? Is it to be labelled adenoma or carcinoma? First of all, let us review the accepted characteristics of these two main groups.

#### *Carcinoma :*

- a. Groups of epithelial cells lying in alveoli.
- b. Cells invade and destroy the tissues of the organ.
- c. Cells invade the surrounding tissues.
- d. Form secondary growths in lymphatic glands and distant organs and tissues.

#### *Adenoma :*

- a. Groups of cells imitate structure of gland from which they arise. The tumour may press upon and injure adjoining tissues, but the cells do not behave in the manner described under (b), (c), (d).

It will be seen that the appendix tumour cells as demonstrated in my series behave in the manner described as (a) and (b). The disappointing results of inquiries into the after-histories of my cases throws very little light on the points (c) and (d).

At this stage of my labours it seemed very doubtful whether it would be possible to come to any definite conclusion as to the innocent or malignant nature of these tumours—tumours so malignant in microscopic structure, yet apparently innocent in clinical behaviour. My own series had failed to prove anything clinically. Then quite unexpectedly a very important piece of evidence was obtained. A microscopic section of secondary growths in the



FIG. 73.—Case 6. Several large collections and numerous small colonies of cells in alveoli with fibrous walls. ( $\frac{2}{3}$  obj., No. 4 eyepiece.)

liver was shown at the Medical Society of London by the President, Dr. Eustace M. Callender. The primary tumour was found in the appendix. Dr. Callender has kindly placed the section and clinical notes of the case at my disposal, and it has such an important bearing on the subject of this inquiry that I feel justified in giving full particulars.

*Dr. Callender's Case.*—Widow, age 53. The patient had an attack of rheumatic fever when 10 years old, followed by 'slight lesion of the heart'. She was always 'too red'. For the past two years attacks of diarrhoea accompanied with abdominal pain occurred. Latterly these attacks became more frequent, and were accompanied by retching and vomiting. On Nov. 15, 1923, she was first seen by Dr. Callender, who found her well nourished, with plenty of subcutaneous fat, profoundly cyanosed



FIG. 74.—Case 6. Colonies of rounded cells in alveoli the walls of which are composed of unstriated muscle. The growth is also invading the subperitoneal fat. (Same magnification.)

and almost pulseless. The abdomen was universally tender, particularly the upper part and right iliac fossa. The patient improved slightly under treatment, but died suddenly on Nov. 18, while sitting up in bed.

Post-mortem examination revealed 'atheroma of coronary arteries and mitral valve'. The liver was much enlarged and filled with malignant growths. The appendix was enlarged to the size of a 'small Tangerine orange' by the presence of a yellowish-white solid growth. The growth had not spread beyond the appendix.

The blood examination showed a marked condition of polycythæmia.

Microscopic examination of the growth in the liver shows masses of cells contained within well-formed alveoli with fibrous walls. Some collections are small and others very large. Necrosis of some of the large areas has occurred and hæmorrhage has taken place into some of the cell masses. Most

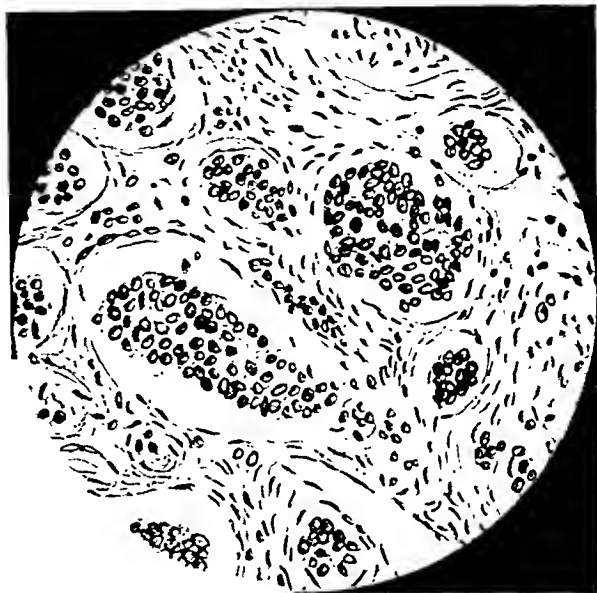


FIG. 75.—Case 6, Colonies of rounded cells closely packed and separated by fibrous bands. ( $\frac{1}{2}$  obj., No. 4 eyepiece.)

of the cells are round in shape and of medium size: the nucleus is small and round. Here and there columnar cells can be seen arranged in imperfect rings and surrounding a small central space (lumen). (See Figs. 77, 78, 79.) A comparison of this picture with those presented by the appendix tumours described above shows them to be identical. There is no room for doubt that we are looking at a secondary deposit of carcinoma in the liver which has originated as one of those curious and mysterious primary tumours of the appendix.

Unfortunately sections of the appendix growth are not available; but in spite of this, it is only necessary to compare the drawings made from the liver growth with those of others from the appendix tumours to make us feel

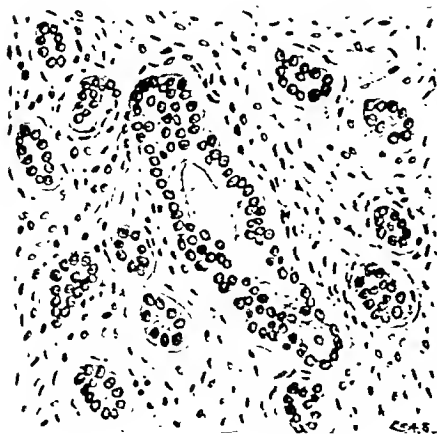


FIG. 76.—Case 6. Note marked tubule in longitudinal section and other tubules in cross-section. The columnar shape of some cells is well shown. (Same magnification.)



certain that we are looking at identical growths. Assuming then, from the above evidence, that one of these primary appendix tumours has proved to have given rise to metastatic deposits in the liver, then the vexed question as to whether they are innocent or malignant must be answered—They are malignant. Further, from the fact that columnar epithelial cells are found in the growths we can definitely place the tumour in the carcinoma group.

The indefinite place which these little tumours have in medical literature, and the uncertainty which exists in the mind of the various authors, is well illustrated in the abstracts quoted below. This uncertainty is due to several causes. In the first place, the tumours are rare, and their discovery in the abdomen may be looked upon as accidental in practically all cases. Again, they appear to be of slow growth; and so, after removal with the appendix, the later history of the cases is lost owing to the well-known difficulty of

keeping in touch with patients after operation. If some means could be devised to make complete records of the patients' lives after removal of the appendix, some estimate could be made as to what percentage, if any, recurred and destroyed life. I imagine very few cases do recur, for in a long experience of post-mortem work I have not seen one. The establishment of a National Museum of Pathology, which I have been advocating during the past two years, would take such a task in hand.

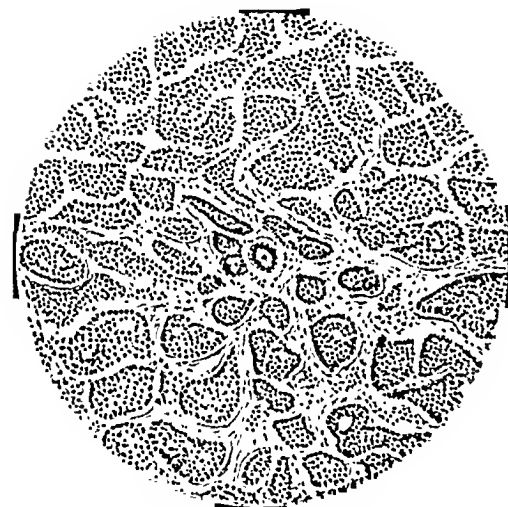


FIG. 77.—*Dr. Callender's case of secondary growth in the liver. The type of cell and general alveolar arrangement of the growth is strikingly like that seen in Fig. 66. There is a small ring of columnar cells in the centre. ( $\frac{1}{2}$  obj., No. 4 eyepiece.)*

It has not been possible to count all the appendices that have passed through my hands during the past twenty years, but as a matter of interest the figures at the Royal Northern Hospital for two years in which they were

examined for another purpose are worth quoting. For the year 1922, 280 appendices were removed by operation, and 209 for the year 1923. These figures are much higher than those of ten or more years ago, but they serve to indicate the rarity of this variety of carcinoma.

### LIST OF CASES.

The following cases are enumerated in chronological order, and are accompanied with such data as were available or considered pertinent to each. Of these cases, five were treated at the Royal Northern Hospital, and six occurred privately.

I wish to thank those gentlemen who have given me permission to use

their cases, and for the trouble they have taken in looking up old records and writing to the patients' medical advisers to obtain later information to make the cases complete. I also wish to thank my friend Dr. W. H. McKinstry for his kindness in looking through the medical literature, and Mr. S. A. Sewell for his excellent drawings.

*Case 1.*—(Figs. 66, 67, 68). May 25, 1904. Male, age 19. (Mr. McAdam Eceles.) Symptoms pointed to some lesion in the region of the appendix, and it was removed.

A small caseating tumour was found in the tip of the appendix. Slight fibrous adhesions were present on the outer surface of the organ.

The patient was last seen two years after the operation and was then quite well.

*Case 2.*—June 21, 1906. Male, age 29. (Mr. Mower White.) The patient had suffered with pain after food for five years. Latterly the pain was continuous. On examination a sausage-shaped tender swelling which could be 'rolled' under the fingers was detected through the abdominal wall. The appendix was removed by operation, and a small bulbous swelling found at the distal extremity. No after-history could be obtained.

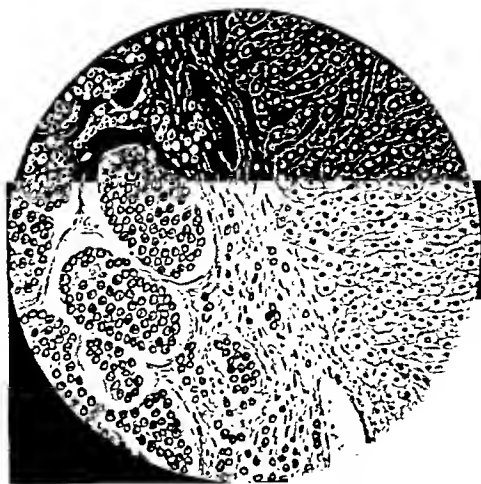


FIG. 78.—Dr. Callender's case. Includes a portion of liver tissue on the right. ( $\frac{1}{8}$  obj., No. 2 eyepiece.)

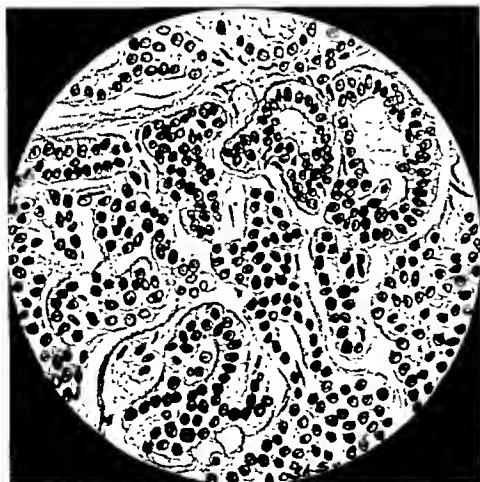


FIG. 79.—Dr. Callender's case. Shows large irregular tubules of columnar cells. The other cells are closely packed and of indefinite shape. The reticulation in the tubules represents extravasated blood. ( $\frac{1}{8}$  obj., No. 4 eyepiece.)

*Case 3.*—(Figs. 69, 70, 71). Sept. 23, 1909. Male, age 59. (Mr. Mower White.) Some time before, the patient had suffered from an abscess about the duodenum. Latterly symptoms of appendicitis developed, and an operation was performed for removal of the organ.

The appendix is short and thick, and its surface covered with old fibrous adhesions. The distal end is rounded off, and on section found to contain a mass of solid growth. The muscular wall can be traced all round the tumour. (See Fig. 71).

No after-history was obtainable.

*Case 4.*—Sept. 12, 1913. Mr. W., age about 35. (Mr. Mower White.) Patient had recently returned from America, where a diagnosis of appendicitis had been made. More recently he had what appeared to be a slight attack of appendicitis, and an operation was decided upon.

The appendix shows a slightly nodular mass at the tip and the peritoneal surface is smooth.

The doctor in charge reports that he has quite lost sight of the patient.

*Case 5.*—June 18, 1914. Miss E. W., age 38. (Mr. Mower White.) History of increasing abdominal discomfort for two years. Pain in right side and back lasting a few hours. Slight tenderness over McBurney's point. Nothing else found.

Bulbous swelling near tip of appendix. Solid yellow material in centre of swelling. (*See Fig. 72.*)

No after-history was obtained.

*Case 6.*—(*Figs. 73-6.*) Oct. 7, 1914. Spinster, age about 50. (Mr. Mower White.) Patient had an attack of abdominal pain, with tenderness and swelling in the right iliac region. There was some doubt as to the diagnosis of appendicitis, and a few high enemata were given. At the time of the operation there was no evidence of peritonitis, and the cæcum was empty.

Large yellow cascating mass at tip of appendix.

Patient died five years later from nephritis. There was no clinical evidence of new growth. No post-mortem examination was made.

*Case 7.*—April 3, 1917. Mrs. M. A., age 54. (Mr. Barrington-Ward.) An abdominal tumour was noticed for six months, accompanied with flatulence, constipation, and frequency of micturition. At the operation a multilocular ovarian cyst was found, and removed together with the appendix.

Appendix acutely inflamed, and its distal end is occupied by a small solid firm mass.

A letter of inquiry was returned through the post.

*Case 8.*—Nov. 19, 1919. Female, age 25. (Dr. D. R.) Operation performed for appendicitis. Appendix enlarged, and its distal extremity is occupied by a solid bulbous mass.

No further record obtainable.

*Case 9.*—March 7, 1923. S. F., female, age 11. (Mr. Mower White.) The patient was brought up to London by ambulance from a boarding school, where she had developed what appeared to be a subacute attack of appendicitis. On arrival in London there was a tender swelling in the right iliac fossa. The appendix was removed the following day. It showed evidence of recent inflammation, and there was definite evidence of local peritonitis.

Appendix about 2 in. long; wall very thick and covered with slight adhesions. On opening the organ an ulcer was found about the middle with thickened mucous membrane around. Microscopically the base of the ulcer was found occupied by new growth, and a small nodule of carcinoma found on either side.

The patient is now in good health.

*Case 10.*—June 9, 1923. Miss E. H., age 63. (Dr. Malcolm Donaldson.) History of metrorrhagia for ten years, with occasional pain in the back and weakness after the attacks. At the operation the appendix appeared nodular, and was removed as a matter of routine.

After removal the uterus was found to contain a soft fungating fibroid in its cavity, and its body was occupied with many hard fibroids. The appendix is very short, and its distal end contains a soft, nodular, and yellowish growth in which tiny calcareous points are present.

The patient was seen in February, 1925, and was in good health.

*Case 11.*—June 2, 1924. Mrs. S., age 60. (Mr. Eric W. Sheaf.) The patient's symptoms pointed to some abdominal lesion, and clinical investigations left some doubt as to whether the gall-bladder or appendix was affected. A laparotomy revealed an appendix showing very marked chronic inflammatory changes, and it was removed. The gall-bladder was found to be normal, and no other lesion was discovered. Short, thick, and white appendix. The tip is bulbous and solid.

The patient is much better since the operation.

The number of cases in the above series is too small to furnish any accurate statistics of age and sex incidence. It is interesting to observe, however, that five patients had reached or passed the age of 50 years. Of the remainder, two were between 30 and 40, two in the twenties, and one patient was only 19 years of age. The youngest of all, a girl of 11, presents two exceptional features—the growth occurred in the body of the appendix and consisted of two nodules. It is worth noting in connection with the last two cases that, although carcinoma occurring in young people usually grows rapidly and soon destroys life, these two patients were alive and well two years after the growths were removed. The sexes are represented by 4 males and 7 females.

## QUOTATIONS AND ABSTRACTS FROM VARIOUS AUTHORS.

These are added in order to illustrate the views held by observers who have investigated the problem as to the nature and origin of this growth.

BOWLBY AND ANDREWS. *Surgical Pathology and Morbid Anatomy*, 1920, 7th ed., p. 553 :—

“Malignant disease may also simulate simple inflammation of the appendix. It is a comparatively rare condition, and is seldom diagnosed before operation. The growth may be a columnar-celled carcinoma, but in several cases which have been recorded the disease has been of a singularly chronic nature, devoid of any tendency to give rise to secondary growths and occurring at an earlier age than is usually the case in true cancer. The microscopic appearances in these cases has been not unlike those seen in endotheliomata of the parotid region.”

BLAND-SUTTON, *Tumours Innocent and Malignant*, 1922, 7th ed., p. 368 :—

“Primary cancer of this small segment of the intestinal tract has been recorded by many observers, but it differs pathologically and clinically from cancer occurring in any other region of the body. It presents itself as a small nodule in the thickened wall of the appendix; the nodule rarely exceeds a cherry-stone in size. Microscopically it consists of nests of spheroidal cells. No special symptoms are associated with these small tumours. Though in structure they mimic cancer, there the likeness ends, for the nodules do not invade adjacent structures, nor recur after removal, nor disseminate. The patients are usually about 30, but these nodules are found in children and adolescents.”

ROBERT MUIR, *Text-book of Pathology*, 1924, p. 456 :—

“True carcinoma may originate in the appendix, but this is very rare. A less uncommon type of growth, however, occurs as a small nodule usually situated towards the free extremity, and presenting an alveolar structure like a cancer. Such a growth may be very minute, and may be accidentally found on microscopic examination. The cells are somewhat small, and are irregularly arranged in closely packed masses in the submucous coat, though extending into the other coats. In cases of this condition which we have seen we have found no evidence whatever of active growth, and this has been the general experience. It is doubtful whether malignant disease develops in connection with the lesion, although this is a possibility. The term canceroid may be suitably applied. The lesion probably represents a collection of epithelial cells dislocated in the process of development, and the condition, as Aschoff suggests, is comparable to the groups of ‘naevus’ cells which are present in pigmented moles of the skin.”

ROLLESTON AND JONES, *Trans. Med. Chir. Soc.*, London, 1906, xxxix. An important paper in which is incorporated the literature up to 1906. The authors make an historical survey and give an analysis of the figures relating to incidence, age, associated conditions, etc. Their cases include growths in other parts of the intestines, both spheroidal-celled and columnar-celled carcinoma.

The following description of the microscopic structure of two specimens examined at St. George's Hospital is given :—

"The arrangement of the growth is alveolar, the cells occupying the alveoli are round and polyhedral, small, and somewhat resembling those of rodent ulcer. There is a scanty margin of clear protoplasm around the nuclei, which stain deeply and show nucleoli. The cells are in close contact and fill the alveoli, but in some alveoli there are one or two clear spaces, apparently due to degeneration of the surrounding cells. The spaces thus produced are more often empty, but may contain spindle-shaped cells like those forming the walls of the alveoli. This vacuolated appearance is a striking feature of the growth. The alveolar walls are delicate, and formed of spindle-shaped connective-tissue cells.

"It may be difficult to determine the starting-point of the tumour . . . but we incline to the view that it is the mucosa which becomes replaced by spheroidal-celled growth and from the cells of Lieberkuhn's crypts."

The authors refer to a case of multiple primary growths of the ileum and jejunum included in their series, and go on to observe :—

"The histological nature is evidently the same as in those spoken of as primary spheroidal-celled carcinoma of the vermiform appendix. Further, the growths in both cases are nearly always benign, and in this respect, it is true, resemble some form of endothelioma more than spheroidal-celled carcinoma."

KELLY AND HURDON, *The Vermiform Appendix and its Diseases*, 1905 :—

"The size of the tumour varied from 5 to 12 mm. in most cases up to that of a pigeon's egg in others."

"The tumour appeared as a firm white nodule fairly definitely circumscribed, but cannot be shelled out, and in places the margin gradually merges into the surrounding tissue."

The authors go on to state :—

"Only a few of the cases of carcinoma of the appendix conform to the usual type of glandular intestinal carcinomata. The majority of cases belong to a less usual type consisting of round, oval, or irregular alveoli filled with small polymorphous cells, having a scanty protoplasm and sharply-stained vesicular nuclei. Mitotic figures may be seen. In but few places is there any evidence of a glandular formation. Generally, however, where the growth is traced to its origin in the mucous membrane a lumen may be detected in one or two of the alveoli, and it is usually possible to trace a direct histogenetic relationship between the tumour and the crypts of Lieberkuhn."

"The tumour shows a distinct local invasive tendency penetrating the sub-mucous and muscular coats with but few exceptions."

ELWELL. "Primary Malignant Disease of Vermiform Appendix", *Quart. Jour. Med.*, 1908. The author divides the growth into columnar-celled and spheroidal-celled carcinomata. In referring to the second group he remarks :—

"If these tumours are spheroidal-celled carcinomata originating in the columnar cells of Lieberkuhn's glands, they should be most actively malignant. The desire

to avoid the discrepancy afforded by the innocent behaviour of tumours so malignant in structure has prompted several suggestions.

“(A) Appearances due to inflammation. (B) Form, position, and structural peculiarity of the appendix. (C) Growths may be adenoma or endothelioma.”

The above quotations serve to show the large amount of time and thought that the various authors have devoted to the study of this curious little tumour. It will be noted that, in the effort to provide a satisfactory answer to the problem as to the real nature of the tumour, the writers have advanced various explanations. Some of these are different from mine, but as my account was written before seeing their articles, I think it best to let it stand. It represents the conclusion arrived at after many years' study of morbid anatomy and careful study of the microscopic sections of tumours. The drawings appended have been made from selected portions of the sections, and will, I hope, serve to illustrate truly and fully the various points which have caused me to arrive at these conclusions.

I am disappointed at my failure to obtain definite information, either from my own series or the medical literature, of the malignant course of any case. The authors whose articles I have quoted have grouped the ordinary columnar-celled carcinoma cases with the spheroidal-celled type. It is impossible, therefore, to prove how many, if any, of the latter have run a malignant course. The impression gathered from their remarks gives one the idea that almost all the cases pursue an innocent course, but some are malignant. In view of the case reported by Dr. Callender, I think we are safe in assuming that this is correct. If it had only been possible to obtain microscopic sections of the appendix tumour in his case, the evidence would have been conclusive.

No doubt the above authors have, as is my experience, been faced with the difficulty of tracing patients after operation, and so have not been able to complete the cases. Another difficulty is the length of time which may intervene before other growths develop after any primary lesion has been removed. To mention but one instance, a man, alive and well twelve years after resection of a carcinoma of the sigmoid. Many other similar cases could be given.

It is impossible to tell the duration of time taken for the development of these appendix tumours, such little bodies as they are, tucked away in a large sac like the peritoneum. The presence of calcareous specks in No. 10 of my series suggests a slow and lengthy course of growth, and the patient's age rather supports this view.

## A CLINICAL STUDY OF ELEVEN CASES OF VESICAL DIVERTICULA.

By R. OGIER WARD, LONDON.

VARIOUS deformities of the bladder are included under this name. Septic paravesical cavities, which have established communications with the bladder, may be called false diverticula. Traction pouches may form as the result of adhesions to neighbouring organs, and the part of the bladder which is not infrequently found in hernial sacs has been considered to belong to the same group; also those pouches, which sometimes persist at the apex of the cavity owing to incomplete obliteration of the urachus, are true diverticula of the bladder. I have no examples of these conditions to report; moreover, they are quite distinct from the diverticula or pouches (*Fig. 80*) which form the subject of this article, and which may be defined as cavities lined with mucous membrane and communicating with the bladder by means of a single orifice, which is sharply defined and of smaller diameter than the pouch to which it is the exit. At its opening there is often a ring of muscle, which is sometimes spoken of as a sphincter—a description that is incorrect; there are usually muscle fibres in other parts of the wall of the pouch, and particularly near its orifice, but these are commonly scanty and never as abundant, or in such uniform layers, as those of the bladder itself; the mucous membrane lining the cavity is continuous with that of the bladder, though it is not often so well formed, and its submucosa is very attenuated; externally the sac is covered by layers of connective tissue fixing it more or less firmly to neighbouring structures. Often there is evidence of past or present inflammation in all the layers.

Mr. W. E. M. Mitchell has kindly examined microscopically specimens of most of my cases. He reports that all the coats of the bladder are represented in the diverticula. The mucous membrane is usually thin, and so is the submucous coat, in which there is often inflammation. The muscular layers vary in thickness, but some muscle fibres are always present. At the orifice these are specially abundant and may be considered as a sphincter, although a well-formed trabeculum would give the same appearance in section.

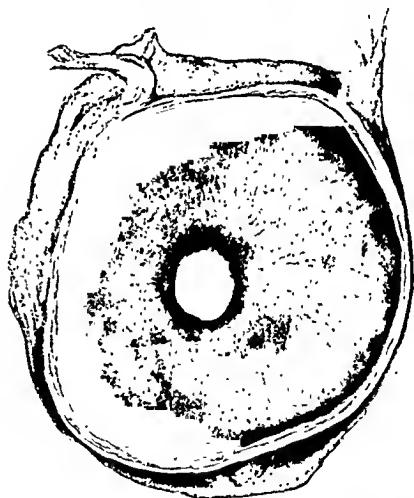


FIG. 80.—The interior of a vesical diverticulum; the opening into the bladder is seen. The fundus has been cut away (operation specimen).

Such diverticula may be single, or there may be two of them sometimes placed symmetrically, or they may be multiple; in which case one of them is often large, whilst the others are of trivial dimensions. They vary in size from that of a hazel-nut to a sac as large as, or even larger than, the bladder itself.

I have tried to ascertain if such diverticula occur in animals other than the human species, and it seems clear that they are very rarely found. Legueu and Papin,<sup>1</sup> in their recent admirable article, quote one case, which has been reported, of bilateral vesical diverticula in a Bengal monkey. But this case is the only example I can find.

Sir John M'Fadyean very kindly writes from the Royal Veterinary College as follows: "I can find no reference to the occurrence of diverticular bladder of any of the domestic animals, and I have never seen anything of the kind myself. There are a good many records of the occurrences of what was called the urachus cyst connected with the bladder; but these are quite clearly due to persistence of part of the urachus after birth".

Sir Arthur Keith, Dr. C. F. Sonntag, anatomist to the Zoological Society, and Mr. W. P. Pyecraft, of the Natural History Museum, also tell me that they have never met with the condition—an important statement, for each of them has a very wide experience of examinations of all sorts of animals.

Vesical diverticula are of considerable importance, for they may be the cause of serious trouble to the patient, and though the diagnosis is sometimes very difficult, they are in most cases easily recognized if suitable methods are employed, and can usually be removed by a properly-planned surgical operation.

Diverticula do not form a large proportion of those vesical diseases which require surgical treatment. In the last ten years 924 operations upon the bladder were performed at St. Peter's Hospital, and only 27 of these were for the removal of diverticula. Therefore 11 male patients, upon whom I have operated for this condition, seem worthy of report, for they serve to illustrate the symptoms met with, and certain of the complications that may arise; also the results of treatment can be examined. Notes of one of these, which is of special interest, are given later; the others are not recorded in detail, but are discussed in the course of the article.

The origin of these diverticula is uncertain; the various views of different authors were recently summarized by Girling Ball,<sup>2</sup> and are very fully discussed in the article by Legueu and Papin. By many it is held that, although they may not manifest themselves till late in life, they are always due to a congenital predisposition. For it is pointed out that they most frequently occur at certain places in the bladder which are developmentally weak, namely just above the ureteric orifices, in this position being often symmetrical: or less commonly just behind the interuretic bar. Certainly, in the course of routine cystoscopies, it is not rare to find small recesses in the mucosa above and behind the openings of the ureter; and these may well be the precursors of true diverticula which develop later in life when some obstruction has occurred to the outflow of urine from the bladder. Another view is that they are formed from accessory ureteric buds.<sup>3</sup>

In the present series of cases there are two examples of solitary diverticula opening close above the ureter, slightly internal to it in one, and in the



other slightly external; two others had similarly situated diverticula with smaller sacculi near them; and there is one example of an orifice immediately behind the interureteric bar, almost in the mid-line. One patient had symmetrically placed diverticula with openings just lateral to those of the ureters; and in two others there were a fully-formed diverticulum on one side and some smaller ones in a corresponding position near the opposite ureter. Thus the situation of the pouches in these eight cases shows that they originated in those parts of the bladder where the musculature is known to be congenitally weak. In two others, the openings were far on the posterior wall of the bladder and far removed from the ureters.

One diverticulum was almost certainly congenital. This was in a boy 15 years old. Cystoscopy showed the trigone well formed except at its left outer angle; here its boundaries disappeared into the opening of a diverticulum, and when this was removed



FIG. 81.—The shadow of the catheter enters the bladder. The diverticulum lies behind, and projects to the right of the picture.

by operation, the interureteric bar was found to be continued into it as a well-marked ridge upon its inner and posterior side. On this ridge, about 1 in. from the bladder, was the opening of the left ureter, which was quite normal in appearance. Outside and rather above the opposite ureteric orifice, which was normal, was a shallow depression in the bladder wall, and in it were several small recesses. Elsewhere, except for a slight degree of trabeculation and some cystitis, the bladder was healthy, and there was no obstruction either at its outlet or in the urethra. A cystogram of this case is shown in *Fig. 81*. The bladder is seen in the middle of the pelvis rising vertically over the shadow of the sacrum. The diverticulum lies obliquely, extending towards the left ischial spine, but being also visible to the right of the middle line, and

it is nearly as large as the bladder itself. This case may, perhaps, be considered a variety of the rare deformity known as double bladder.

When diverticula occur apart from any obstruction to the outflow of urine from the bladder they must certainly be congenital, and the boy is probably an example of this, though one cannot exclude the possibility that at some period of life a defect had existed in the neuromuscular mechanism controlling the emptying of his bladder. But even when obstruction is present, it may well be argued that this has merely made obvious the presence of a pre-existing pouch by causing a great increase in its size, or perhaps an infection of its contents. In illustration of this possibility there may be mentioned two patients who gave histories of old strictures which had been treated at

intervals for many years, and five who were proved to be suffering from enlargement of the prostate, which was obstructing the emptying of the bladder. In two others, although the prostate was slightly enlarged, the removal of the diverticula was sufficient to reduce the residual urine to less than 1 oz.; and since cystoscopies carried out one year later showed no further development of sacculation, it is assumed that the obstructive factor here is a small one, and will prove too slight to effect further changes in the remainder of the bladder.

It is well known that vesical diverticula are decidedly uncommon in women, who are, of course, much less liable to interference with the proper emptying of the bladder than are men, and although from time to time one sees shallow sacculi near the ureters in the female bladder, yet well-formed diverticula in the female are comparatively rare. This must give force to the view now generally held, and supported by the study of these patients, who were all males and, except for the boy, all between forty-four and sixty-eight years of age, that although diverticula of the bladder can be entirely congenital in origin, and occur apart from any obstruction, yet such cases are much less common than those in which obstruction is present. And this, though often of slight degree, is, when added to a congenital predisposition, an important cause in the production of the majority of diverticula large enough to give rise to symptoms. It must be stated, however, that true diverticula can also result from obstruction alone, and an illustration is afforded of this by one patient who had suffered from a narrow urethral stricture for some years, with marked difficulty of micturition and attacks of retention. When the bladder, which was much distended, had been emptied, its wall was more than  $\frac{3}{4}$  in. thick; multiple diverticula were present, three of which were larger than hens' eggs, and had an unusual amount of muscle in their structure. Their orifices were on the posterior and postero-lateral aspects of the bladder, well away from the areas in which the musculature is normally somewhat defective; and the smaller sacculi, which were very numerous, opened, for the most part, the same regions. The multiplicity of these pouches, their disposition, the presence of severe urethral stenosis, and the very marked hypertrophy of the bladder, make it exceedingly probable that they were an acquired condition entirely due to the greatly increased intravesical pressure, and not associated with any pouches of congenital origin.

### SYMPTOMS AND COMPLICATIONS.

The more moderate-sized diverticula do not, as a rule, give rise to *symptoms* unless they become infected. The larger diverticula, even before infection occurs, usually give the patient sufficient discomfort to cause him to seek medical advice. The symptoms observed in these eleven cases will now be examined, and it will be seen that they are often due to some *complication* which has arisen.

The boy of 15 would not have consulted his doctor had not an infection of his bladder and *hematuria* occurred, for until he was 13 he considered himself perfectly well in every respect; then he began to get pain, referred to the end of the penis during micturition, with some urgency; sometimes

he would be free of this for a month or two, but he gradually got worse. He had no difficulty in micturating, and the stream was normal, except that he noticed that the urine first passed was often turbid, though it became clearer as the bladder emptied. Slight frequency of micturition was present, five times by day, and once by night. He took little heed of these symptoms, however, and only became interested when, on several occasions during the last six months, he passed blood intimately mixed with his urine. When examined, his urine contained some blood and was turbid from the presence of pus. Cystoscopy showed generalized cystitis, also the diverticulum which has been described. As the left ureter opened within it, only the right ureteric orifice could be seen through the cystoscope. Presumably the hæmaturia was due to inflammation of the diverticulum or of the bladder, for no other cause could be found; and since the operation, a year ago, there has been no recurrence. One other patient had suffered from intermittent hæmaturia for seven months, which was the result of inflammation. It was never severe, though occasionally small clots were passed, and removal of the diverticulum has cured it completely.

But the commonest cause of hæmaturia associated with this abnormality is not uncomplicated inflammation, but the presence of one or more *calculi* in the bladder or diverticulum. It is not surprising that *inflammation* should occur, and that calculi should form; for though the wall of the sac usually contains muscle fibres, yet this musculature is always inferior in development to that of the bladder itself. The ring of muscle, which is often seen around the orifice, has only some of the functions of a sphincter, and these only in a very imperfect degree. Therefore, when the patient micturates, the bladder empties itself in two directions, namely, through the internal urinary meatus into the urethra, and through the partly contracted orifice into the diverticulum. When the bladder is empty the diverticulum sometimes discharges its contents, but usually the act is very incompletely performed, and probably, in every case, some urine remains in it, even if special postures are adopted to encourage drainage by gravity, and, indeed, even after catheterization. This fact can sometimes be demonstrated by a cystogram taken after the patient has been allowed to void as much as he can of the opaque fluid, for it will show the diverticulum to remain partly filled. These conditions of stagnation are ideal for the occurrence of cystitis and the formation of calculi, the diagnosis of which may sometimes be difficult.<sup>4</sup> One case, in which such a complication was present, is worth describing in detail from this point of view.

The patient was a man who in 1923 was aged 48; when eight days old he had been so crudely circumcised that most of the skin of the penis had been removed, and an acquired glandular hypospadias produced, with stenosis of the urethral orifice. He had been under treatment at St. Peter's Hospital since 1907, and the urethra had been gradually dilated to size 13/15 English. In 1914 cystoscopy showed general cystitis, normal ureteric orifices, with clear effluxes, and there was generalized trabeculation. The stricture contracted when he did not attend for treatment, and gradually his cystitis got worse, the urine becoming very foul, gravel was passed, and hæmaturia occurred on many occasions.

In 1923 I found that he had 4 oz. of very offensive residual urine, and stones could be felt in the bladder. Through the cystoscope a rather poor view was obtained of an intensely inflamed bladder and a calculus in it. In June he was admitted to hospital, and an X-ray (*Fig. 82*) showed three calculi to be present. In

spite of the fact that it could be seen that the stones were not in contact with each other, I wrongly assumed that they were all in the bladder, and performed litholapaxy. As this did not cause much bleeding, I cystoscoped him before he left the table, and though the visibility was far from perfect, no calculus or fragments could be seen in the bladder. His symptoms soon recurred, and, in October, cystoscopy showed two calculi in the bladder, which was much inflamed and very irritable. Litholapaxy was again performed; on this occasion, after the calculi had been crushed and evacuated, another could be felt with the lithotrite, but could not be gripped by it. A radiogram taken a few days later showed this calculus clearly, and through a cystoscope it could now be seen partly projecting from a diverticulum, which opened above the left ureteric orifice. There were also some shallow sacculi above the right ureter.

Further treatment had unfortunately to be postponed until April of the next year. By this time another calculus had formed in the bladder. At operation, the stone in the diverticulum was found to be tightly fixed in it, with a mushroomed surface the size of a shilling extending into the bladder cavity.



FIG. 82.—Calculi in the bladder and in a diverticulum.

The faults of diagnosis and treatment are well exemplified here. X rays not infrequently show more calculi than have been visible by cystoscopy, for some may be hidden by those nearest to the internal urinary meatus; but when one or more of these shadows are seen to be remote from the others, the probability is that these calculi are outside the bladder itself. Such calculi are shown in *Fig. 83*, which is an X-ray of a patient who had suffered from a stricture for many years, and also an enlarged prostate, and who, it may be mentioned, had, like the other cases now being considered, recently passed blood in his urine.



FIG. 83.—One large and one smaller calculus lying in symmetrically placed diverticula. The faint shadow of a vesical calculus is also seen.

At operation, a soft calculus, probably chiefly composed of blood-clot, was found in the bladder. This is seen as a faint irregular shadow in the centre of the pelvis; two well-formed calculi were also found, each lying in symmetrically placed diverticula, the larger being on the right side. These are clearly seen in the radiogram to lie so far from the position

usual for bladder calculi that they must be outside its cavity, and therefore, having regard to the patient's history, certainly to be calculi lying in diverticula. But even if the appearances are not so obvious, it is sound

practice to have further X-rays taken after litholapaxy, if cystoscopy beforehand has not clearly excluded such possibilities.

Another, but much less frequent, cause of hæmaturia is the presence of a *new growth* associated with a diverticulum, or in rare cases growing within it. I have only one example to report (*Fig. 84*). The diverticulum, which contained a small friable calculus, opened high up on the posterior wall of the bladder, and was about the size of a bantam's egg. Close to its orifice was a spheroidal-celled carcinoma, slightly larger than a hazel-nut, which extended almost up to it, whilst by its extension through the bladder wall it had attached itself to the exterior of the pouch. Hæmaturia had first been noticed five years previously, and not again until twelve months before he reported for investigation. Since that date it had recurred frequently. Though there was slight enlargement of the prostate, which was the cause of some frequency and difficulty of micturition from which he suffered, the bleeding was clearly from the growth, and the soft calculus had formed in blood-clot. After operation there was no more hæmorrhage.

Hæmaturia, in association with vesical diverticula, may be due, therefore, to inflammation, to the presence of calculi, or to a new growth; and it may,

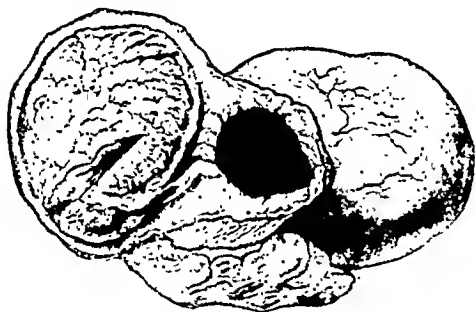


FIG. 84.—A vesical diverticulum with a carcinoma close to its orifice (operation specimen).

of course, be due to the coincidence of an enlarged prostate. One of my cases had suffered from it intermittently for four months; there were several shallow sacculi and a small diverticulum, which only admitted the terminal phalanx of the index finger. His prostate was enlarged, and was the cause of the bleeding and of the presence of 4 oz. of residual urine, for after prostatectomy, without removal of the pouch, the bladder emptied completely and hæmaturia ceased.

Hæmaturia is a warning which a patient does not often allow to pass unheeded, but it is, of course, not so common an event in this disease as is the occurrence of *troubles of micturition* of various kinds.

'*Miction en deux temps*' is often considered pathognomonic of vesical diverticulum, but it must be remembered that it sometimes occurs in tabetic affections of the bladder and urethra, and in a lesser degree in enlargement of the prostate. However, it is a very important sign and strongly suggestive of this condition, but it should be clearly understood that this is not a common symptom. Only one of my cases presented it, and it was not invariably present; he had a large diverticulum (*Fig. 80*) holding more than half a pint of urine, opening behind the interureteric bar, almost in the middle line, with a smaller one beside it.

*Frequency* of micturition is often met with, and this common symptom is usually to be explained by the cystitis which so often results from the presence of the pouch; in other cases it may be due to an enlarged prostate.

*Difficulty* of micturition, in these cases, often results from the association of an enlarged prostate, or stricture, but may be produced by the diverticulum itself; one patient had suffered from this in a marked degree for four months, but said he could overcome it entirely if he micturated whilst lying on his back. The diverticulum opened above and behind the right ureteric orifice; it was as large as the bladder itself, containing probably more than 15 oz. of urine, and extending deeply into the pelvis (*Fig. 85*); on one occasion *retention* of urine had occurred; these troubles were entirely removed by its excision. The explanation of retention in this disease which most writers have offered, and one which would account for it, at any rate, in most instances, is that the presence of such a diverticulum interferes with the proper action of the trigonal musculature, one of whose functions is to open the internal urinary meatus at the commencement of the act of urination. Two other patients had also had retention of urine, presumably from the same cause. The first had two diverticula, one the size of a walnut, and one not larger than a hazel-nut; both opened near the left ureter. The other was the man whose bladder musculature was so greatly hypertrophied and who had multiple acquired diverticula. Even after two of the three largest had been removed, and a urethral stricture excised, retention persisted.

*Pain* is a symptom usually present when inflammation has developed, and four cases of this class presented it in the form typical in cystitis. One patient, however, who had severe cystitis, would not admit that he had ever suffered from it. Four others, who were free of all inflammation, were also free of pain.

There remain two whose pains cannot be explained by cystitis, but must be directly attributed to the presence of a diverticulum, which in each instance was a large one. The first was the man previously mentioned, who had occasionally 'miction en deux temps', and it was the onset of this pain more than the frequency and slight difficulty of micturition which caused him to consult his doctor. It was related to urination, occurring just before urine commenced to pass from the urethra, being really severe and felt particularly in the left groin, and to a much less degree in the perineum. It ceased as soon as micturition was finished. This was probably due to the forcing of urine into the diverticulum, and the stretching of the adhesions which, as is so often the case, surrounded it.

The other was the man who, as previously stated, suffered from difficulty in passing urine except when in the supine position. Fifteen months previously

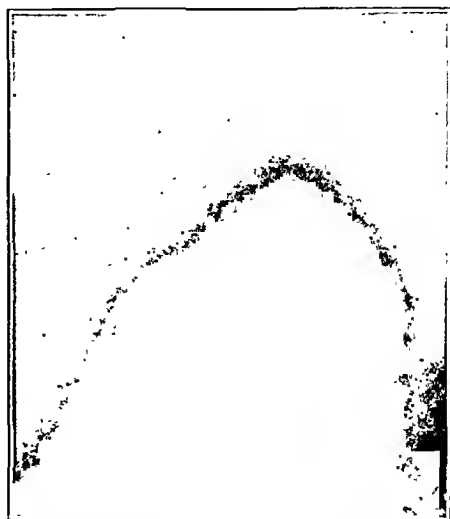


FIG. 85.—Cystogram. The bladder shadow is spherical, and below the diverticulum lies obliquely, and extends higher up.

he had at times experienced pain in the urethra during urination, but this had passed off. The pain of which he chiefly complained, and which he considered a more serious part of his malady than the attack of retention of urine which had once affected him, existed for a year and a half, and took the form of an aching in the region of the left posterior superior iliac spine, which spread down the posterior aspect of the left thigh, and caused him to be lame; he was, in fact, suffering from left sciatica. The diverticulum, as has been said (*Fig. 85*), was almost as large as the bladder, and lay behind the left lower part of the bladder, extending deeply beneath the peritoneum into the pelvis, and being in contact with its left lateral and posterior boundaries. Since its removal the neuritis has disappeared entirely. He attributes the cure directly to the operation, and it seems reasonable to agree with him, for it is probable that the diverticulum was in contact with the coverings of the sciatic nerve in the pelvis, and certainly there were adhesions in this region.

In addition to the complications which have already been discussed, and the possibility of *rupture* of the sac, two others, which are less common, may be mentioned. Firstly, *compression of a ureter*, for this duct is often in close contact with the sac, and from this may result hydronephrosis and the risks of renal infection, though it did not happen in any of my cases. And, secondly, *constipation* from pressure upon the rectum. One of the two patients suffering from very large diverticula complained of this, for his bowels had always acted regularly until recently, when it became necessary, after passing a motion, to defæcate again an hour or so later.

### DIAGNOSIS.

The most important method of diagnosis available in this condition is, of course, *cystoscopy*. If a good view is obtained, the opening of the pouch with its sharply-defined margins is seen as a black hole in the bladder mucosa. unless, indeed, it is only a shallow pouch, in which case its walls are usually visible. The appearance is very characteristic, and easily distinguished from the small sacculi so commonly seen in the trabeculated bladder; but unless the cystoscopy is a thorough one, and all parts of the bladder are systematically examined, it is quite possible that, even if no special difficulties are encountered, the opening might escape observation, although most of the diverticula have their openings near the ureters, which is usually the region most closely inspected. Sometimes a diverticulum can be palpated through the abdomen, but not unless it is a very large one, or takes origin in the upper parts of the bladder. More often they can be felt on rectal examination as a smooth elastic tumour above and behind the prostate, but it would, of course, be unwise to base a diagnosis on this finding without confirming it by cystoscopy, since several other conditions need to be excluded. Through the cystoscope not only can the opening be seen, but a *ureteric catheter* can be passed into the diverticulum, and an estimate thus formed of its size. Sometimes, but not often, the cystoscope passes into the diverticulum. This happened in the case of the man who had sciatica; and the glistening whiteness of its relatively anæmic wall contrasted well with the yellow pinkness of the more vascular bladder mucosa.

The size of a pouch can be best estimated by means of a *cystogram*. The bladder and diverticulum are filled with opaque fluid through a catheter. It is very useful to watch this under a screen, and, when it is completed, to take photographs in whatever positions are best calculated to show the form and extent of the pouch. (Fig. 86.) Other photographs should be taken after the catheter has been allowed to drain, or the patient allowed to micturate. (Fig. 87). Radiograms should also be taken apart from cystography, for, as has been seen, they may reveal calculi whose existence would otherwise not be suspected.

Simple *catheterization* does not necessarily give any indication of the contents of a diverticulum, for the reasons that residual urine may also be



FIG. 86.—Cystogram showing a small diverticulum as a small circular shadow imposed on that of the bladder.



FIG. 87.—Same as Fig. 86, catheter withdrawn; micturition has emptied the bladder but not the diverticulum.

present in the bladder itself, and also that very frequently it is quite impossible to empty a diverticulum by any other means than swabbing it out, or emptying it by suction through the bladder opened suprapubically. Fortunately, however, the other means at our disposal already mentioned, namely, the use of the cystoscope with a ureteric catheter, and the radiographic methods of examination, enable us to determine, in all but quite exceptional cases, the presence or absence of a bladder diverticulum, together with its position, and to form a fairly accurate estimate of its size.

It may be well to point out that it is a serious matter to overlook the presence of a diverticulum in dealing with a case of enlarged prostate, or of any other form of urethral obstruction; for operation will not be followed by relief of symptoms and restoration to health if a diverticulum of any considerable size is also present and not removed. On the contrary, pyuria



will persist, and disorders of micturition, though perhaps somewhat diminished, will continue to exist.

### TREATMENT.

It is because vesical diverticula cannot diminish, but must inevitably increase in size, and because infection so very commonly occurs, that the proper treatment is to excise them.

**Palliative Treatment.**—If, for any reason, an operation cannot be performed, palliative treatment must be tried with the object of encouraging drainage of the contents into the bladder, and so diminishing sepsis. The outlook is not promising, because irrigations through a catheter really only cleanse the bladder itself, for the reason already stated, namely, that these pouches are unable to get rid of their contents because of the lack of competent musculature in their walls, and often because of the position of their orifices. The best form that palliative treatment can take is to encourage the patient to find some position in which he can perform 'miction en deux temps', and, when he has effected this, to wash out his bladder through a catheter, repeating the process several times. Such methods are not without danger, and are obviously a very imperfect form of treatment.

**Operative Treatment.**—The operative treatment of vesical diverticula has been very fully described in a recent paper by Swift Joly.<sup>5</sup> He points out that a preliminary cystostomy should be avoided if possible, since it drains only the bladder, and makes it more difficult than ever to wash out any of the contents from the diverticulum, since the bladder cannot now be distended, and therefore the irrigations fail to pass through the closed orifice into the pouch. If the sepsis is too great to risk immediate excision of the diverticulum, or if the patient's renal efficiency is so poor that preliminary bladder drainage is essential, then, as he suggests, it is best to pass two tubes through the bladder and the opening of the diverticulum into its cavity, so that the irrigations be circulated thoroughly through it. Marsupialization of the pouch has been recommended for such cases, but is not so good an expedient. Any system of drainage should be used for as short a period as possible, because of its manifest imperfections.

The *excision* of a vesical diverticulum is always an interesting and sometimes a difficult operation. The space in which the operator has to work is confined, and the freeing of the fundus of the sac from the pelvic floor, as is often necessary, demands good illumination, and that the patient should be in the Trendelenburg position. I think that such a posture is not good for old men, and should be used for as short a time as possible. It is only this, and the necessity of disturbing severely the attachments of the pelvic peritoneum, that make the operation a severe one, for otherwise there is little shock, and bleeding is comparatively slight.

It is convenient to begin by mobilizing the distended bladder as widely as possible, usually without opening the peritoneum, though, if it is opened on purpose or accidentally, it should be closed before the bladder is incised. This should be done at the stage when it is decided that a finger inside the diverticulum will facilitate the dissection of it from the surrounding tissues. It should be emphasized that careful dissection is the important part of the

operation, and must be carried out with precision and under good observation ; otherwise there may be troublesome bleeding from the veins on the pelvic wall, and there is real danger of damaging a ureter ; for this structure is always in close relationship with a large diverticulum, or one of any size arising from the lower part of the bladder, being either on its posterior wall or lying in the recess between it and the bladder. A catheter in the duct during this part of the operation is helpful, but even this will not prevent its injury if the dissection is not carried out under view. It is the close relationship of the ureter which constitutes one of the chief dangers of operating entirely from within the bladder, and attempting, by volsellum forceps or other means, to invert the sac into it. Other obvious risks, which make such a proceeding thoroughly unsound, are possible damage to peritoneum or bowel ; it is, moreover, entirely unnecessary. Small diverticula, and especially those that lie deep, may sometimes advantageously be removed by a transvesical operation, in which the orifice is encircled by an incision through the mucous membrane and the other coats of the bladder wall, and the diverticulum gradually dissected from its outer attachments whilst it is being drawn like a detached and empty glove-finger into the bladder ; one of the operator's fingers inserted into it may help in effecting this. But such a procedure depends for its success upon the careful external dissection of the sac, and by this means it is possible to avoid injury to other structures which may well follow attempts to turn it inside out by force,

However, the procedure that will be found most generally useful is, as has been said, to mobilize the bladder widely before opening it ; next, to empty it, and to continue the freeing of the diverticulum with the help of a finger inside it ; then, as suggested by Swift Joly, to incise the bladder down to and around the opening of the pouch, so that in the last stage of the dissection it is free from attachment to the bladder. The gap in the wall of the latter is then sutured, and one drainage tube is usually placed outside it for a few days, and one into the bladder cavity in the manner usual after cystotomy.

It is well to remember that operations in the pelvis are never easy in the presence of obesity. Apart from this, the difficulties vary with the position of the pouch, those extending deeply into the pelvis requiring special care ; and also with the amount of adhesions produced by inflammation ; often a plane of cleavage is found in which the dissection of the sac progresses easily, and in any case it is wise to keep as close to the latter as possible. In the absence of sepsis no harm will result if some small part of the fundus of the sac is left in the pelvis, and it may sometimes be well to rest content with this result.

Diverticula were excised from nine patients, and all survived the operation. Two months later one who had quite recovered from this died, after a prostatectomy, from a pulmonary embolism on the third day. The other eight are alive and cured.

It is important to realize that in this disease it is very often insufficient merely to excise a diverticulum, for an obstruction should always be suspected until its non-existence can be proved. No obstructive factor was present in the case of the boy of 15, and he was completely cured of his symptoms by the excision of the pouch ; it was, of course, necessary to reimplant the left

ureter into the bladder, but, as far as can be seen one year after the operation, the kidney has in no way resented this, and the patient is quite well. However, in four of the older patients, from whom diverticula were excised, prostatectomy was necessary for their complete cure. In one of these there was very obvious enlargement of the gland: this was the man who died. In two others, however, rectal examination showed no enlargement at all; nor did cystoscopy reveal any; and indeed it only became clear that there was such an abnormality when the suprapubic wound would not close. In one case, this was at length effected by tying a catheter into the urethra, but two days later the wound reopened and would not heal again. In each of these cases an adenomatous prostate, no bigger than the normal gland, was then enucleated, and uninterrupted healing of the wound led to a speedy and complete cure.

In the fourth example—the man who had a vesical carcinoma as well as a diverticulum—a small adenomatous prostate, recognized at the first cystoscopy, was removed for increasing symptoms one and a half years later.

In two patients the pouches were not excised; one of these, from whose bladder and diverticula calculi were removed by cystotomy, was submitted to prostatectomy three weeks later, and nearly a month after this operation he died of bronchopneumonia. A post-mortem examination showed the bladder almost completely healed. In the other, prostatectomy only was performed, and the diverticulum, which admitted the terminal phalanx of a finger, was left *in situ*, and it seems unlikely that its removal will become necessary.

#### NOTES ON THE YOUNGEST PATIENT IN THIS SERIES.

V. E., age 15. Apparently a healthy boy, at work, and playing games. For two years had suffered from pain after micturition, referred to the end of the penis, with occasional intermissions for two or three weeks. The urine first passed had been turbid; towards the end of micturition it became clear. Six months ago, and on several subsequent occasions, hæmaturia had occurred, the blood being intimately mixed with the urine and never in very large amounts. Usually had to get up once at night to pass urine, and did so five times by day; occasionally frequency had been more marked. The stream passed was normal in size and projection, and there had never been any difficulty in micturition.

ON EXAMINATION.—He appeared perfectly healthy, except that his urine contained pus and blood and a small amount of albumin. Cystoscopy showed the opening of a diverticulum at the left outer angle of the trigone, occupying the normal position of the left ureter, which could not be seen. The right ureter was normal, and so was the rest of the bladder except for some cystitis and a slight pouching of the bladder above and external to the right ureter, and here were two small and shallow recesses.

OPERATION.—The bladder was exposed suprapubically and freed from the peritoneum: it was then opened. The diverticulum, which was unusually thick, was found to be lying very deeply in the left side of the pelvis, from which position it was freed by the dissection of its exterior. With the help of a finger inside it, the left internal iliac vessels and the vas were exposed, and there was a good deal of bleeding, which, however, was easily controlled.

The bladder wall was then incised down to and around the orifice of the diverticulum, and on raising this the left ureter was seen entering its posterior aspect. It was cut across and implanted again into the upper part of the lateral incision in the bladder wall, since it could not easily be brought down to the usual position of entry. The bladder was drained, and a smaller tube was placed outside it to drain the pelvis.

The wound was sutured in the usual manner. The operation, including induction of anæsthesia, took nearly two hours, and the patient stood it well.

Recovery was uneventful. One year later the patient reported that he had been perfectly well ever since the operation. The wound was well healed, and the urine normal. There was no longer any pain on micturition, or frequency by day or night.

### CONCLUSIONS.

1. Vesical diverticula may be entirely congenital in origin, but usually obstruction is associated, and causes them to become large enough to produce symptoms.
2. The commonest symptoms result from infection of the stagnant urine in the pouch.
3. The cystoscope and cystogram provide accurate means of diagnosis.
4. Excision is necessary for cure, and the results are good.
5. When obstruction is present, it also usually requires operation.

I have much pleasure in acknowledging my indebtedness to Mr. Kenneth Walker, who kindly provided me with three cases for operation, to Mr. W. E. M. Mitchell for his report upon the microscopic appearances of the operation specimens, and to Mr. Thornton Shiells for his drawings of operation specimens.

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**POLYPI IN THE BOWEL CAUSING INTUSSUSCEPTION.**

By W. E. M. WARDILL, NEWCASTLE-ON-TYNE.

THAT polypi in the bowel are frequently seen at operations for intussusception is a matter of common experience, but I venture to suggest that the relationship between the two conditions is ill-understood and may give rise to errors

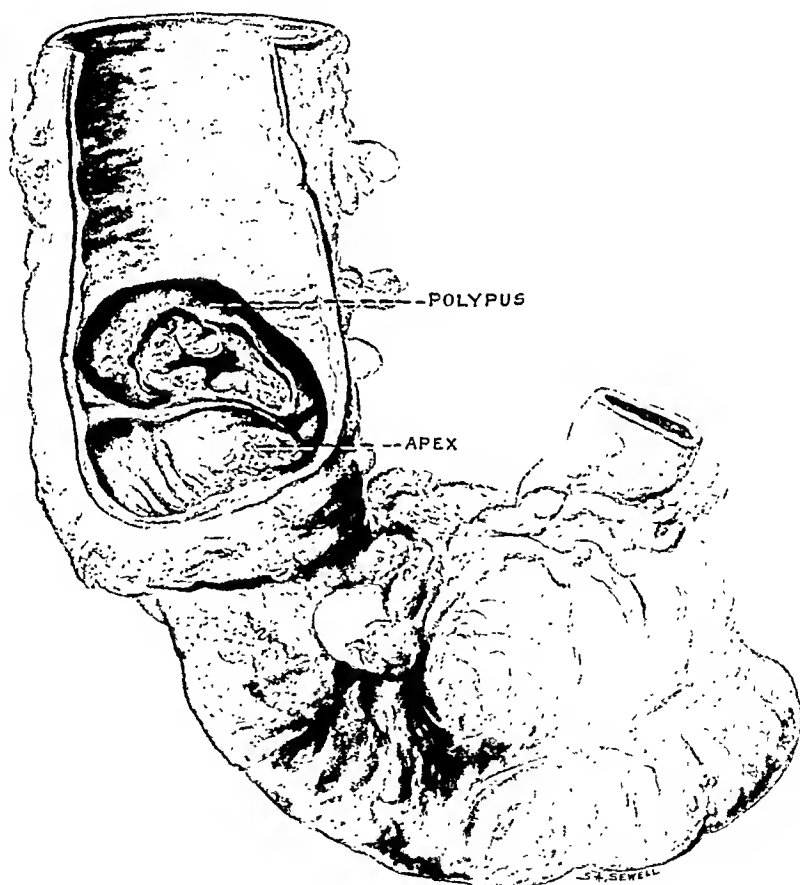


FIG. 88.—Submucous lipoma at the apex of a colic intussusception.

of omission on the part of the surgeon. The pitfalls of abdominal surgery are sufficiently numerous, and the scope of this note is to point out yet another.

One always assumes that when a polypus of the bowel is associated with intussusception there is a relationship of cause and effect. Such, no doubt,

is the case. The polypus is usually regarded as being dragged along by the peristaltic movements, and its point of attachment being thereby invaginated and forming the starting-point or apex. *Fig. 88*, showing a polypoid submucous lipoma of the colon at the apex of an intussusception, is illustrative

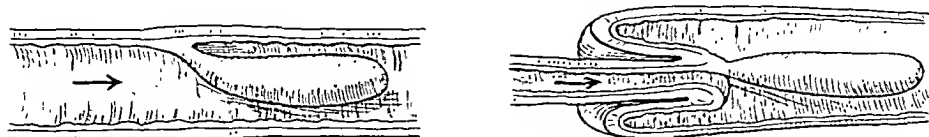


FIG. 89.—Diagram illustrating the mechanism of production of the intussusception shown in *Fig. 88*.

of this, and the diagrams (*Fig. 89*), are intended to explain the mechanism of its production.

But that this simple explanation does not account for all cases—one might even say the majority—is amply proved by reference to many operation and

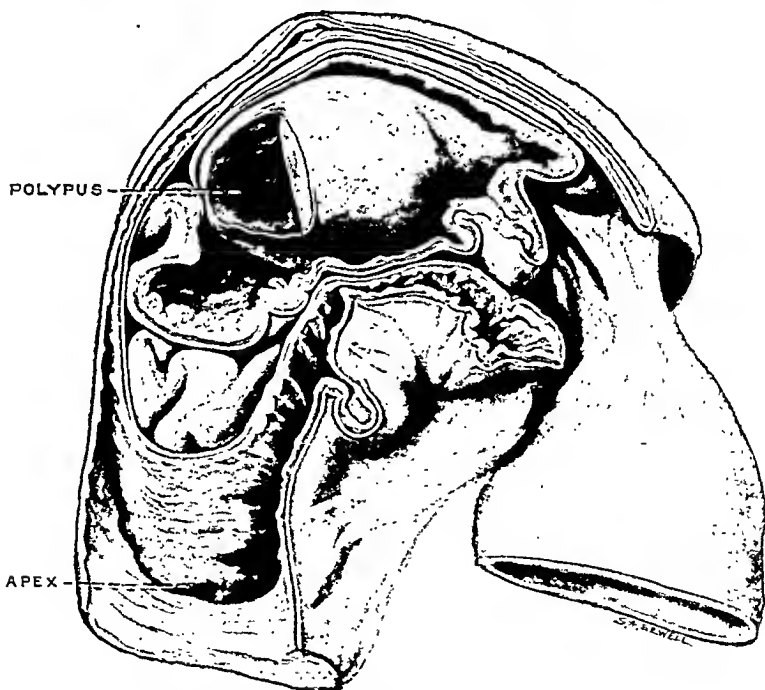


FIG. 90.—Submucous angioma of the jejunum with intussusception. The tumour lies some distance proximal to the apex.

museum specimens. *Fig. 90* is drawn from a specimen of intussuscepted submucous angioma of the jejunum. The tumour in this case occupies a position which is some considerable distance proximal to the apex, although it is still included in the intussusceptum. *Fig. 91* depicts an intussusception

caused by an inverted and polypoid Meckel's diverticulum, previously reported by Mr. Hamilton Drummond.<sup>1</sup> Here, again, the attachment of the polypus is some distance proximal to the apex of the intussusception. A fourth specimen (*Fig. 92*) shows an enteric intussusception associated with an adenomatous polypus, which is not even included in the intussusceptum, but lies far on the proximal side. This specimen is of considerable interest, because it represents the recurrence of an intussusception after an operation for the same condition some years previously.

It is obvious from the examination of these specimens that mere traction upon the polypus by intestinal movements does not account for the formation of the intussusception. The probable explanation is that the tumour, lying

within the lumen of the bowel, acts as a foreign body and produces spasmodic contraction of the gut around it, with inhibition of that part immediately distal. The conditions are now favourable for that final act of peristaltic gymnastics whereby the contracted part is induced to slip into the dilated portion (*Fig. 93*).

Lindsay and Perrin<sup>2</sup> have adduced evidence to show that the intussusception of infants is associated with inflammation of the lymphoid tissue in the lower ileum, and they offer this as an explanation of the age incidence. Such an explanation is welcome, since it obviates the necessity of taking refuge in terms like perverted peristalsis, where the bowel is expected to display suicidal tendencies for no apparent reason. Moreover, it puts the intussusceptions of infancy on the same footing as those associated with polypi and growths. If

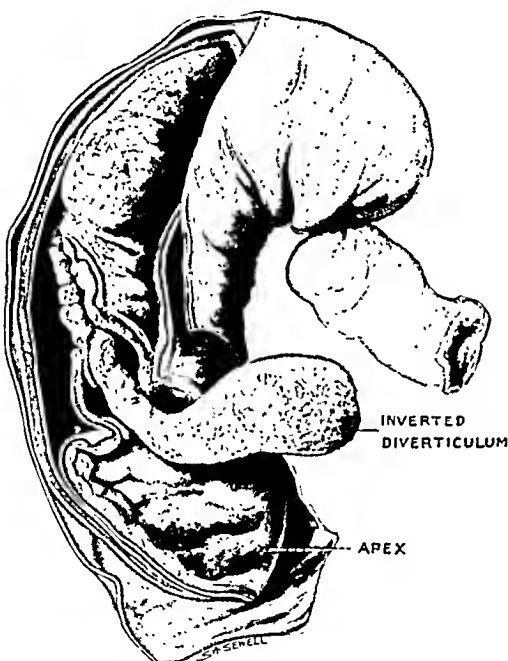


FIG. 91.—Inverted Meckel's diverticulum with intussusception. The attachment of the diverticulum is proximal to the apex of the intussusception.

the explanation given by myself of the relationship between the polypus and the bowel is accepted, it is easy to see the mechanism of production of the ileocecal type of intussusception. No dragging of the bowel wall by the foreign body need be assumed; the terminal part of the ileum simply contracts violently and is pulled by its longitudinal muscle into the caecum, which is relaxed ready to receive it. Once an apex is formed, the intussusceptum itself becomes the foreign body, and attempts are made to expel it, with the result that more and more invagination takes place. The same mechanism which produces the first intussusception is capable of producing a second and forming a double intussusception, and, theoretically, this process might continue until the bowels were like the familiar series of

boxes within a box. There is a limit to this, however, in the distensibility of the ensheathing layer.

The moral to be drawn from the above observations is that, when operating for intussusception, particularly in subjects over the age of two

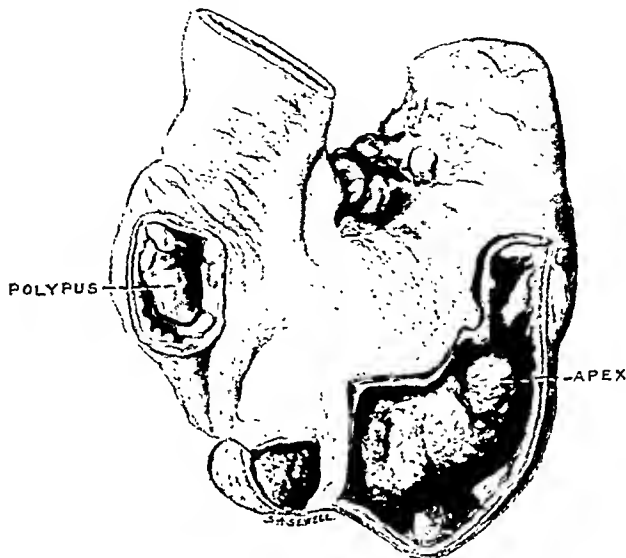


FIG. 92.—Adenomatous polypus of the small intestine causing recurrent intussusception. The tumour lies in a proximal position, and is not even included in the intussusceptum.

years, careful examination should be made of the proximal healthy bowel for possible polypi. Neglect of this precaution may result in recurrence, immediate or remote, as illustrated by the specimen shown in *Fig. 92*. Knowledge of this relationship helped me in the following case, of which I give short notes.

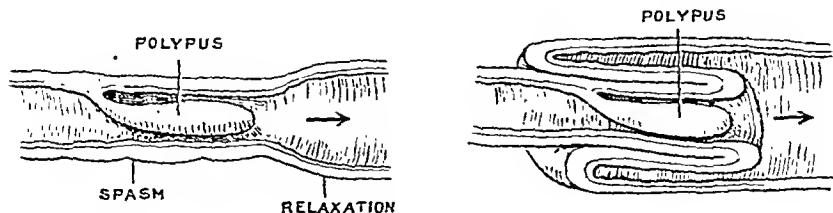


FIG. 93.—Diagram illustrating the usual way in which a polypus starts an intussusception.

A man, age 43, had suffered for seven months from intermittent abdominal pain. During the last fortnight the pain became more severe, and he had difficulty in getting his bowels moved. Two days before admission to hospital the pain became very bad, and he passed bright-red blood with his motions. When I saw him, he complained bitterly of agonizing pain, and demanded



relief at any cost. The abdomen was slightly distended, and there was a small amount of free fluid. A mass, somewhat obscured by the distention, was felt in the situation of the descending colon and was thought to be growth. At operation, this turned out to be an intussusception of the colic type. It was apparently of recent origin, and was easily reduced, there being no adhesions. After reduction, the bowel was carefully examined for growth, but none could be felt at the site of the intussusception. Some four or five inches proximal to this, however, there was a small hard carcinoma confined to one wall of the bowel; it had no relation to the intussusception, and had one not been on the look-out for a possible tumour in this situation it might easily have been overlooked, and the abdomen closed without the true nature of the disease being recognized. The condition of the patient did not warrant further interference, so a safety-valve œstostomy was performed as a preliminary to future operation.

Three weeks later, the abdomen was opened again for the purpose of removing the growth. On examining the site of the disease, the intussusception was found to have recurred, and on this occasion the growth was actually in the intussusceptum at the apex. There was no œdema of the bowel, and the appearances suggested that recurrence had taken place, at the earliest, a few hours before operation. The diseased bowel was removed, and anastomosis done by the method of Fraser and Dott. Recovery was uninterrupted.

The drawings in this note are from specimens in the Durham University College of Medicine, Newcastle-on-Tyne.

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# **INTERSCAPULO-THORACIC AMPUTATION FOR CARCINOMA OF BREAST.**

By SIR CUTHBERT WALLACE, K.C.M.G., C.B., LONDON.

ON two occasions recently an interscapulo-thoracic amputation was performed for advanced carcinoma of the breast.

The problem was to provide a covering for the raw surface left after wide removal of the breast. In the ordinary fore-quarter amputation the line of union between the anterior and posterior flaps runs on the outer side of the thorax. In an amputation for carcinoma of the breast, the line of union must be at the sternum, for it is necessary to sacrifice the anterior flap which

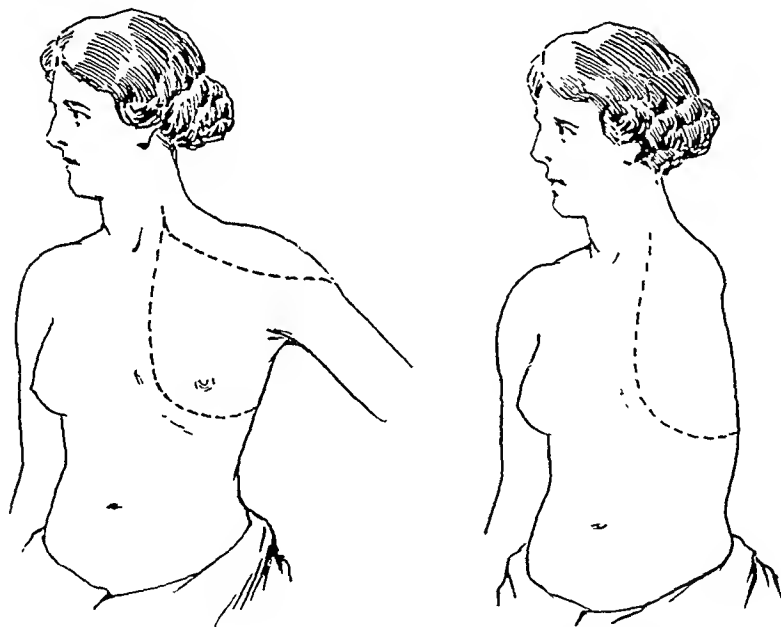


FIG. 94.—Interscapulo-thoracic amputation for carcinoma of breast.

contains the tumour. The skin to cover the front of the chest must come from the posterior flap, which thus has to reach the sternal edge.

In planning the incision in the first case, it did not seem possible that the posterior flap could reach so far, and preparations were made for an extensive graft taken from the forearm of the amputated limb. The result showed, however, that a posterior flap could be fashioned that would reach the sternal edge.

In the second case advantage was taken of the experience gained in the first, and the contour of the flap was somewhat modified, with the result that accurate and easy coaptation of the skin was obtained.

The incision started over the sternomastoid, about 3 in. above the clavicle, passed down to and over the sternoclavicular articulation; it then turned outwards 2 in. below the clavicle, crossed about 2 in. above the angle formed by the junction of the skin of the chest and the arm; then across the deltoid, sloping slightly downwards to the point where the posterior aspect of the arm joined the body; then backwards and downwards almost to the angle

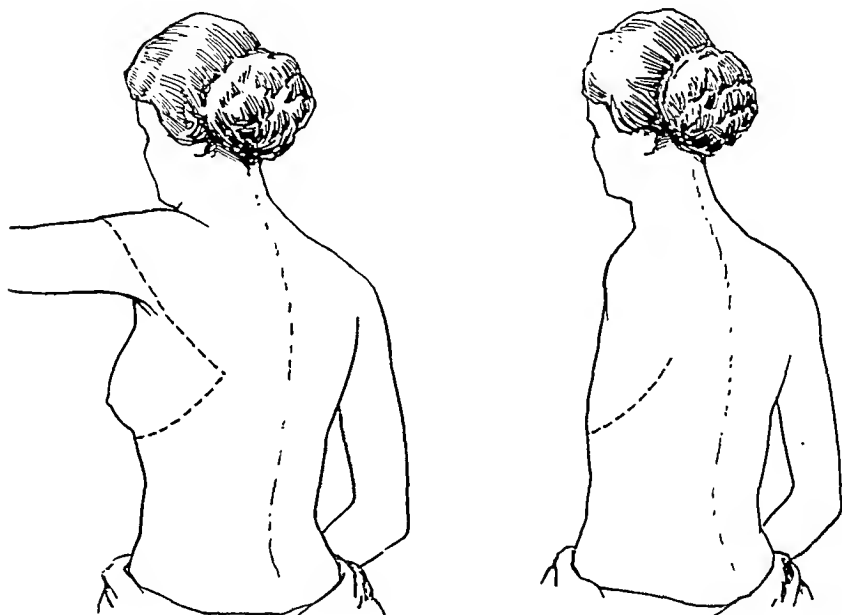


FIG. 95.—Back view of operation shown in Fig. 94.

of the scapula. From this point it turned forwards, crossed the axilla horizontally below the border of the mamma, and reached the sternal edge, which it followed to the sternoclavicular articulation.

By turning the superior flap upwards, it was quite easy to divide the muscles on the superior and vertical border of the scapula and turn forwards the upper extremity and detach it from the thorax. The continuation of this process enabled the arm, scapula, pectoral muscles, and mamma to be removed in one block. (*Figs. 94, 95.*)

The shock was no greater than that encountered in many breast operations.

In each case a small part of the anterior edge of the skin-flap necrosed, but otherwise the flaps healed well.

## *SHORT NOTES OF RARE OR OBSCURE CASES.*

### TWO CASES OF POLYPUS OF THE STOMACH.

BY T. B. MOUAT, SHEFFIELD.

THE following two examples of mucous polypus of the stomach illustrate the complications which may be set up by this relatively infrequent condition, for in both a partial inversion of the stomach wall had resulted from the drag of the pedicle of the tumour, which had been extruded from the stomach into the duodenum. In the first case obstructive symptoms were present, and the distended duodenum had perforated, while in the second the polypi showed malignant changes, and one of them had become ulcerated and given rise to hæmorrhage from the stomach. In the absence of complications, the diagnosis would appear to depend on the presence of a palpable tumour in the pyloric region, which



FIG. 96.—Case 1.  
Mucous polypus of stomach.

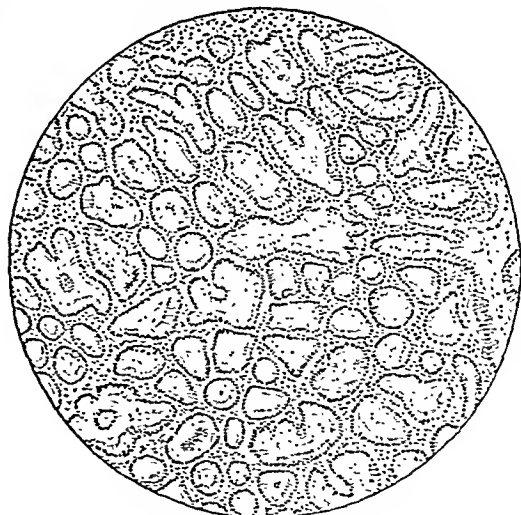


FIG. 97.—Case 1. Pen drawing of slide.

gives a characteristic rounded and sponge-like filling defect in the radiograms of the stomach after a barium meal.

*Case 1.*—Harriet T., age 12, was admitted to the Royal Infirmary, Sheffield, on June 16, 1924, suffering from diffuse peritonitis. For the past twelve months she had been troubled with recurring attacks of epigastric pain and vomiting, which lasted for three or four days. Between these attacks she appeared to be in quite good health. The vomit was described as being "just like chopped grass". The present illness commenced six days

ago with the same symptoms, which continued till the day before admission, when the pains grew very acute, and she became collapsed.

ON ADMISSION.—The patient appeared a fairly well-grown but thin girl, with a pinched, dusky face and sunken eyes. Pulse 135. Temperature 100°. Generalized abdominal rigidity, with some distention, and signs of free fluid in the peritoneal cavity.

OPERATION.—On opening the abdomen through a right paramedian incision, gas and a copious purulent odourless exudate escaped. The incision was enlarged upwards, and adherent lymph was seen on and around the

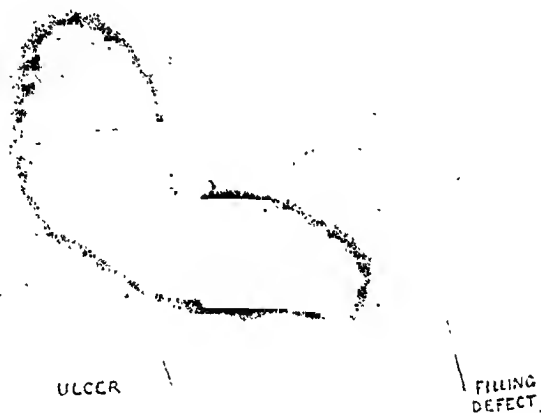


FIG. 98.—Case 2. Radiogram of stomach.

duodenum. The pylorus and the first part of the duodenum were distended, and on grasping these segments of the gut to pull them down for closer inspection, a large mass was felt to slip from the duodenum back into the stomach. It was then noticed that the perforation lay concealed under the duodenal attachment of a rounded fleshy band which ran between the under surface of the liver and the anterior aspect of the first part of the duodenum, and appeared to have resulted from stretching of old adhesions. This band was removed, and the leaking perforation, which was round in shape and about one-eighth of an inch in diameter, was invaginated.

The stomach was now opened, and the tumour, which proved to be a large mucous polypus attached by a short pedicle to the posterior wall of the

stomach near the lesser curvature, was removed with a small portion of the mucosa at the site of attachment. The gap in the mucosa and the incision in the anterior wall of the stomach were sutured. The purulent exudate in the peritoneal cavity was swabbed out, and the pelvis drained through a separate suprapubic 'button-hole' incision.

The after-progress was uneventful; the drainage tube was removed on the second day, the patient went home with both wounds healed in the fourth week, and remains in good health.

The gross appearance and microscopic structure of the specimen are shown in *Figs. 96 and 97*.



FIG. 99.—Case 2. Resected portion of stomach, showing malignant ulcer of the greater curvature and two polypi in the pyloric antrum.

*Case 2.*—Thos. W., age 40, a colliery corporal, was admitted to the Royal Infirmary, Sheffield, on Sept. 25, 1924. His mother had died from cancer of the stomach at the age of 47.

In excellent health till about the end of June, 1924; he then began to be troubled by a dull pain in the pit of the stomach, which became worse after food. The appetite was poor, and he lost a stone and a half in weight. He stopped work on July 23, and four days later vomited a washhand-basinful of blood. The pain became extremely severe, and he sent for his doctor

(Dr. Ritchie, Hoyland Common, Barnsley), who kept him in bed and gave him medicine. The pain lasted for about a week, but there was no recurrence of the vomiting, and since then he has gradually felt better. He attended hospital as surgical out-patient on Aug. 18. A rounded tumour about the size of a golf ball could be felt in the pyloric region, and he was advised to come into hospital for operation. On Sept. 22 he came back to the hospital to tell us he felt so well that he wanted to resume work; but as the tumour could still be felt in the epigastrium, he was dissuaded from doing so, and was admitted on Sept. 25.

Radiograms of the stomach after a barium meal showed a filling defect at the pylorus, and an oblique ulcer niche on the greater curvature (*Fig. 98*).

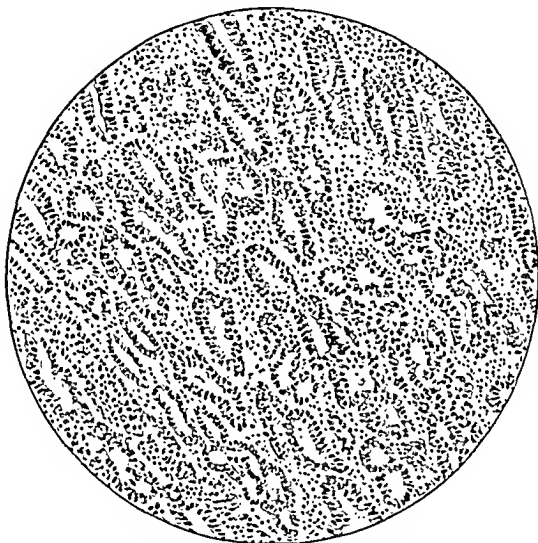


FIG. 100.—Case 2. Pen drawing of slide.

OPERATION, *Sept. 29*.—An indurated and obviously malignant ulcer was found on the greater curvature about three inches from the pylorus. The pylorus was greatly enlarged and appeared to be stretched over a rounded mass which protruded into the first part of the duodenum, and which slipped back into the stomach when the parts were handled. A partial gastrectomy was performed by Pólya's method, the stump of the stomach being brought down through the mesocolon after completion of the anastomosis. The patient made an uninterrupted recovery, and left hospital at the end of the third week.

The resected portion of stomach (*Fig. 99*) shows on the greater curvature the oblique ulcer cleft, which appears to have resulted from breaking-down of a malignant nodule, and two polypi, which are attached to the walls of the pyloric antrum. The edges of the ulcer and the polypi showed well-marked malignant changes, and the pen drawing (*Fig. 100*) shows the structure of the less malignant portion of the larger polypus.

# INTUSSUSCEPTION OF THE SMALL INTESTINE EXTRUDED THROUGH A FÆCAL FISTULA ON THE SURFACE OF THE ABDOMINAL WALL.

By E. T. C. MILLIGAN, London.

THE patient was a boy, age 11, with a fæcal fistula resulting from an enterostomy performed six months previously by another surgeon to relieve post-operative intestinal obstruction following acute appendicitis for which the child had been operated upon seven days before. At the operation for intestinal obstruction, the first presenting coil of distended intestine—which proved to be 1 foot proximal to the ileocaecal junction—was selected for the enterostomy. The child developed scarlet fever a few days subsequently,



FIG. 101.—Intussusception of small intestine extruded on the surface of the abdominal wall.

which delayed the closure of the enterostomy. The abdominal wall became very red and ulcerated by the action of the intestinal contents.

**PRESENT ILLNESS.**—On July 10, 1924, six months after the previous operations, the child suddenly experienced an attack of abdominal pain and vomiting. The pain recurred several times. In the intervals of freedom from pain the child was quiet, but pale in appearance from shock.

When operated upon, about nine hours after the onset, there was presenting on the abdominal wall about 8 in. of small intestine intussuscepted and



extruded through the faecal fistula. The intussuscepted bowel consisted of entering and returning layer, there being of course no ensheathing layer, so that the mucous membrane of the bowel was presenting. The intussusception was curved, bluish red, and congested from constriction at the point of exit from the abdomen, and bled readily on manipulation.

Attempts at reduction failed owing to rigidity of the bowel and constriction at the skin opening of the fistula.

OPERATION.—The scar surrounding, and the fistulous opening in the abdominal wall, were excised, the abdomen being opened by prolonging the resulting wound upwards and downwards. Resection of the intussusception was then performed and an end-to-end anastomosis made. The intussusception seemed to have started about 4 to 5 in. proximal to the fistula. No cause was discovered. In spite of the unhealthy condition of the skin of the abdominal wall, healing occurred by first intention, and recovery was complete and uneventful.

*Fig. 101*, which Mr. Thornton Shiells has made from rough drawings at the time and from description, is an excellent picture of the appearance at the operation.

## CASE OF VON RECKLINGHAUSEN'S OSTEITIS FIBROSA.

BY A. RENDLE SHORT, BRISTOL.

Miss D., age 46, was sent to me by Dr. Ward, of Wotton-under-Edge, suffering from pain and swelling of the right knee.

HISTORY.—As long as she could remember there had been some bony swelling about the knee and also of the fingers of the right hand; she suffered from fits between the ages of 20 and 30. Six years ago, after an injury, there was synovitis of the knee, which cleared up; eighteen months ago the fluid returned without obvious cause, but again gradually cleared up. Four months ago the joint swelled again and has not improved.

ON EXAMINATION.—The patient is small-made but intelligent. General health is good. There is no thyroid tumour palpable. There are a number of large naevo-fibromata on the chest, abdomen, and right hand, of very long duration. The right knee is very swollen, with soft irregular bosses arising from the tibia and the femur; there is no fluid present. It is very tender and painful. The right hand shows, in addition to the naevo-fibromata, bony swelling of the phalanges, especially at their bases; on account of the deformity two fingers were amputated many years ago. The phalanges are apparently involved in the same disease as the knee. The radius and ulna on the left side are curved, but not expanded. In spite of this, the right arm is shorter than the left.

X-RAY REPORT.—The skiagram (*Fig. 102*), taken by Dr. Mayes, shows extensive changes in the bones entering into the knee-joint. The femur shows a thin shell of hard cortex, enclosing a nearly uniform transparent tissue which extends for several inches up the shaft. Here and there one can see

rounded or elongated denser areas. In front, the cortex has been perforated by the transparent mass, which forms a projecting tongue-like swelling, folded down on to the shaft. The upper end of the fibula shows a similar condition, but it has not extended beyond the limits of the bone. Both the femur and the fibula are slightly expanded. The tibia shows a more localized transparent area, which has bulged out through the cortex both behind and in front. The



FIG. 102.—Osteitis fibrosa : skiagram of knee.

patella retains a hard shell in front, but the rest of the bone is transparent. There are a few dense pearl-like shadows of bone in the capsule of the knee-joint, and the articular surface of the tibia is irregular.

In view of the extensive nature of the disease in the femur and tibia, operation was thought to be contra-indicated, and a walking caliper advised.

Three weeks later the patient was sent to me again on account of great

pain in the knee, inability to put the leg to the ground, swelling. The joint was found to be much more swollen. extend it. It was held semi-flexed, and there was but little that very painful. The softish boss over the head of the tibia it did not show eggshell crackling. The fingers were un-

OPERATION.—As the patient was very averse from excision of the knee was performed; altogether about 7 inches of bone were removed. The bony ends were brought together and fixed by a Hallopeau plate, and Parham and Martin bands. There was good healing, and the plate and bands were removed later. At the time the patient was sent home with the bones ununited, but

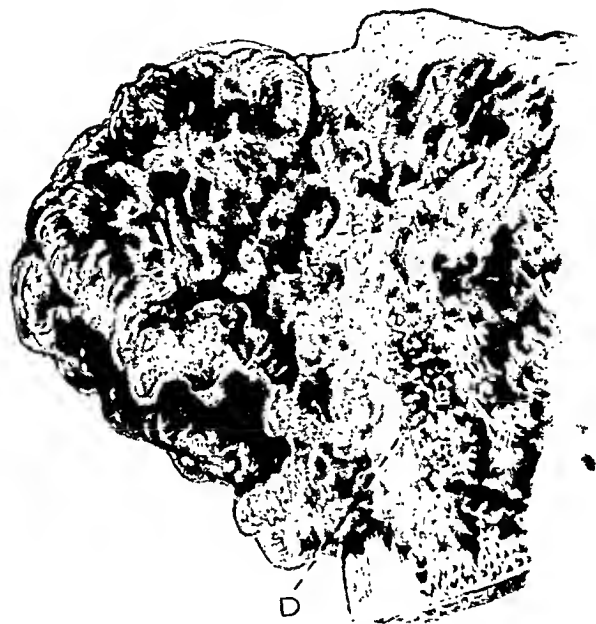


FIG. 103.—Osteitis fibrosa. Section of tibia through  
B, Tubercle; C, Fibula; D, Osseous mass.

comfortably on crutches with the leg in plaster. I put it to the ground on account of the great shoe.

DESCRIPTION OF THE REMOVED BONES.—The removed bones are shown in Figs. 103 and 104. Judging from the diseased area in the tibia was excised, but by the failure of the femur. However, the cut surface of the failure to unite suggests that extensive disease was,

The tibia was the more grossly diseased. A tubercle was removed from the upper three inches of the tibia; a little renal tubercle. The rest had been expanded from within and was very soft. The articular cartilage remained, but was very thin. The material occupying the head of the bone was

with small pearls and rods of cartilage-like material. It varied in colour from greyish-pink in the centre to a red jelly inside the boss at the back of the tibia that had been so painful. Here it was almost diffuent. The fibula appeared normal.

The femur showed less extensive changes. The articular cartilage was thinned and studded with little nodules. The cortex of the bone, though very thin, was intact except just above the patella, where a reddish jelly-like material bulged through, forming a soft swelling like that at the back of the tibia. Elsewhere the femur was completely occupied by a material which had the colour of ordinary bone-marrow in that situation, but softer. No cancellous bone was visible. There was a rod of cartilage-like material lying loose in the middle, well shown in the picture (*Fig. 104*), and some other

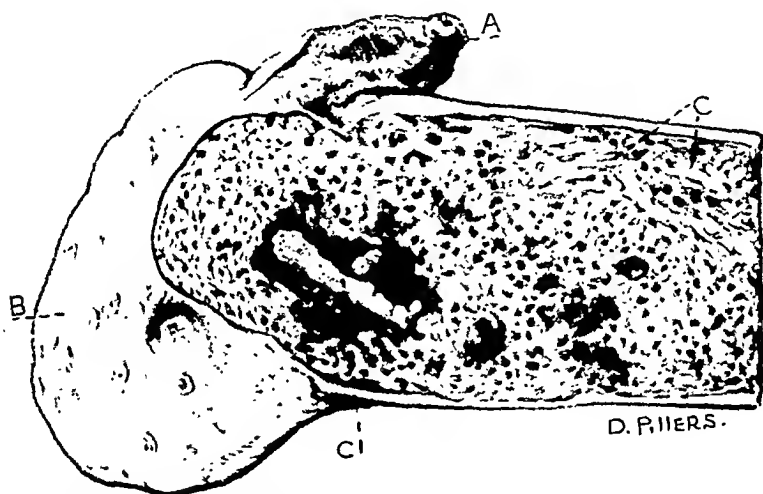


FIG. 104.—Osteitis fibrosa. Section of femur through condyle. A, New growth; B, Eroded surface of condyle studded with nodules; C, Rods and pearls of osseo-mucin looking like cartilage.

areas with little nodules of the same. The substance occupying the femur had not the gelatinous appearance of that found in the tibia.

**MICROSCOPICAL REPORT.**—Professor I. Walker Hall reports: The tissue shows chiefly the later changes associated with osteitis fibrosa. There is generalized replacement of the bony structure by fibrous tissue. The formation of thin-walled capillaries has been followed by numerous hæmorrhages. The attempt to lay down new bone is evident in the strands of osseo-mucinic and osteoid tissue, but the process has not proceeded very far.

The removal of the bone trabeculæ appears to have been brought about more fully by halisteretic atrophy than by osteolysis and osteoclasia. Osteoblasts and osteoclasts have taken a part which is less pronounced than in some cases. They are present in medium numbers only, and do not present the intensive giant-cell formation hitherto described.

## DISCUSSION.

The case is a fairly typical one of von Recklinghausen's osteitis fibrosa, but with some peculiar features.

The various conditions which are grouped under the name of 'osseous dystrophies' have recently been subjected to a careful examination and critical discussion by Dawson and Struthers in a most valuable paper, which draws largely on the extensive researches of von Recklinghausen. The conditions which come under their examination, and enter into the list of possible diagnoses in this case, are: (1) Osteitis fibrosa, generalized or localized; (2) Paget's osteitis deformans; (3) Osteomalacia; (4) Rickets; (5) Myeloid sarcoma, better called 'benign giant-celled tumour' (Ewing).

They point out that the histological features of osteitis fibrosa are an erosion and expansion of the bone with the laying down of new bone on fibrous strands which become converted into osseo-mucin. The Haversian spaces, and later the bone-marrow, are converted into fibrous tissue, with proliferated endosteal cells and, where the bone is being eroded, many giant cells (osteoclasts). There is a tendency to myxomatous degeneration, and cysts, large or small, are plentiful. In addition to these there are tumour-like masses which, both to the naked eye and histologically, may resemble myeloid sarcoma. In their case there was an adenomatous tumour of the parathyroid gland.

In Paget's *osteitis deformans*, the changes are fundamentally very similar, but the giant-cell areas and cyst-formation are absent, and the bone changes are more in evidence.

In *osteomalacia*, there is a simple halisteresis or dissolving out of the mineral salts, leaving zones of decalcified osteoid tissue. There is little or no fibro-osteoid-tissue replacement of the marrow and Haversian spaces, and there are few or no osteoclasts.

In *rickets*, of course, the main change is the exaggerated and irregular cartilage-formation with deficient calcification.

In *myeloid sarcoma*, better called 'benign giant-celled tumour' (Ewing), there is a definite tumour-formation, full of giant cells which are osteoclasts, not myeloplaxes. Osteoclasts have many nuclei; the myeloplaxes or megakaryocytes of bone-marrow show a single many-lobed nucleus.

In the present case, the very long course, dating from childhood and perhaps from birth, the involvement of many bones and, until recently, the freedom from symptoms, point to osteitis fibrosa. The tibia shows bone erosion and expansion; the femur shows a mixture of bone erosion and new bone-formation within the shell of cortex. The marrow has been converted into fibrous and myxomatous material, and shows in places many proliferated cells, probably endosteal in origin. There is not the thick layer of very porous bone one sees in osteitis deformans. The reddish jelly-like area projecting from the head of the tibia closely resembles a myeloid sarcoma, and could certainly be described as a 'tumour-like mass'.

The peculiarities of the case are the following: (1) The association with many subcutaneous nævo-fibromata; (2) The absence of fractures; (3) The great pain, and the swelling of the knee-joint; (4) The scantiness of osteoclasts

in the sections examined (it may be that examination of other parts of the diseased area might show them); (5) The presence of rods and pearls of osseo-mucin in the region which should have been occupied by bone-marrow, looking to the naked eye like cartilage.

## OSSIFYING CHONDROMA OF A RIB MISTAKEN FOR A SARCOMA.

By CECIL P. G. WAKELEY, LONDON.

THE interest in this case is of a threefold nature: (1) As to whether or not trauma can be the predisposing cause of chondromata; for in this case there was a very definite history of injury seven years before the growth became noticeable. (2) Because there was marked pulsation to be felt in the upper part of the tumour, with some involvement of the lower cord of the brachial plexus, and the clinical diagnosis first made was that of sarcoma. (3) This diagnosis was supported by the report of the radiologist, namely: sarcoma of chest wall obscuring the scapula with a certain amount of ossification.

The following is an account of the case: Mr. W. M., age 42, while in France in 1916, was a machine-gun ammunition carrier. In October, during an engagement, he was carrying a loaded ammunition-drum under his right arm, when suddenly it exploded, causing him severe pain in the right side of the chest; otherwise he was not hurt in any way. The pain disappeared during the rest of the engagement. Soon after this incident the patient noticed a pain in the back of his shoulders which was dull and constant. In November, 1916, he developed nephritis and was sent home to England. The nephritis cleared up, but the pain in the back still persisted, so that he had to give up playing tennis. In August, 1923, one morning while drawing his hand across his chest, he noticed a smooth lump about the size of a walnut; it was hard and immovable, but was not attached to the skin. It was situated an inch above and external to the right nipple. He saw his doctor, who told him it was cancer. A month later he was X-rayed at Swansea and told he had a malignant tumour, and was sent to London for radium treatment. Radium was inserted into the tumour in three



FIG. 105.—Skiagram showing chondroma growing from rib. Definite areas of ossification can be seen.

situations. No relief was obtained, and the tumour continued to increase in size. The patient complained of pain and weakness on the inner side of the right forearm, and numbness and tingling in the ring and little fingers. In November, 1923, he had great difficulty in writing, and in February, 1924, had to give up using a pen. He was advised to have an amputation at the shoulder-joint, but he refused and returned to his home. Early in May, he was sent to King's College Hospital with the definite request that deep X-ray therapy should be given to the tumour. A skiagram was taken (*Fig. 105*) which demonstrated an ossifying chondroma growing from the chest wall.

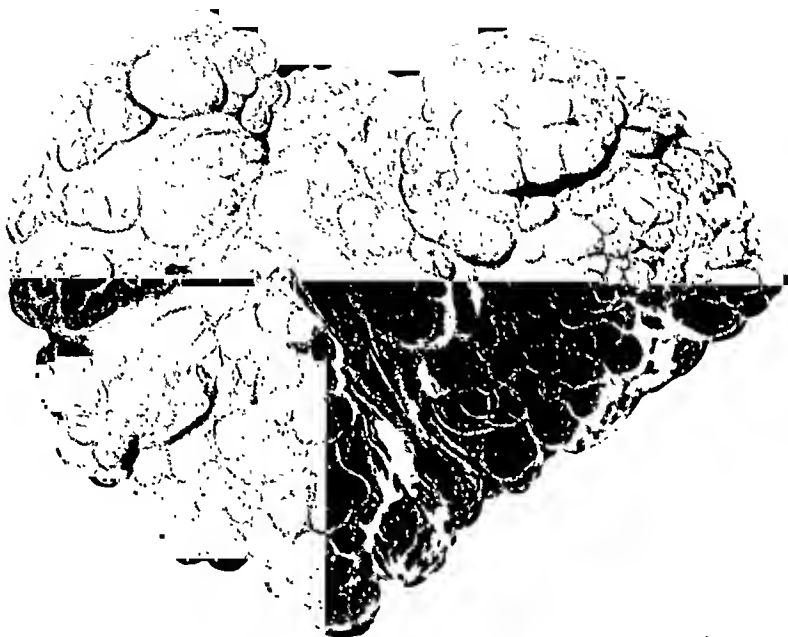


FIG. 106.—Appearance of the tumour after excision.

ON EXAMINATION.—The patient was seen to be a well-built man of medium height. On the right side of the thorax a large rounded tumour was visible; there was definite pulsation at the upper part. The tumour was hard, irregular, and fixed to the chest wall; the pulsation appeared to be due to the axillary vessels which were no doubt stretched over it. There was slight weakness of the muscles in the forearm and hand which were supplied by the lower brachial-nerve cord. The skin over the tumour was stretched and slightly reddened; there were two small healed incisions where the radium had been inserted. The muscles of the thenar eminence were slightly wasted, but all the muscles reacted well on electrical stimulation. The patient was advised to have the tumour removed, to which he consented.

OPERATION, June 15.—A curved incision was made below the tumour, the lower costo-sternal fibres of the pectoralis major muscle were divided, and the tumour was exposed. In separating the fibres of the pectoralis major from the upper part of the tumour, the first part of the axillary artery and vein and the lowest cord of the brachial plexus were found to be lightly stretched over the tumour, and were separated from it with difficulty. The tumour was fixed to the middle of the fourth rib by a short pedicle of about  $\frac{3}{4}$  in. diameter; this was removed, together with a portion of the rib from which it was growing. The periosteum was separated from the rib previous to removing the latter. The fibres of the pectoralis major were drawn together with

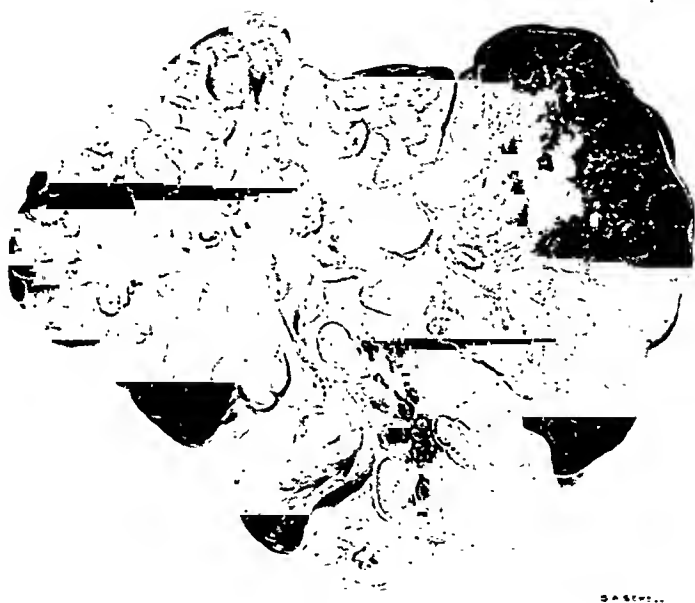


FIG 107.—Sectional view of excised tumour.

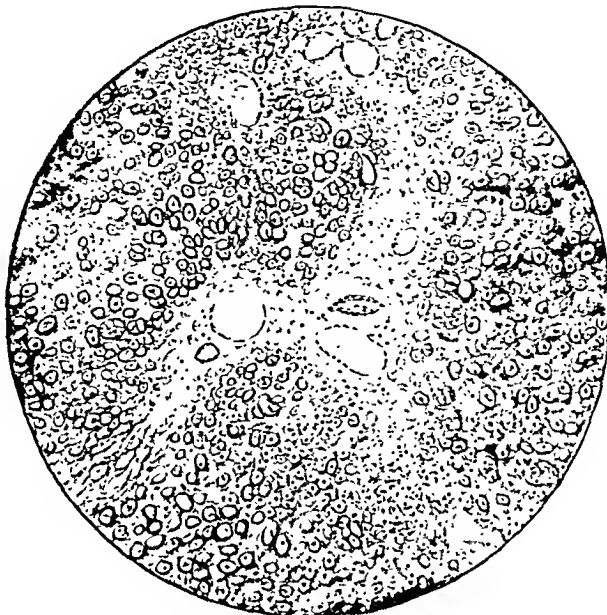
catgut sutures, and the skin was closed by interrupted silkworm-gut sutures; a large drainage tube was inserted for twenty-four hours. The wound healed well, and the patient was discharged from hospital at the end of June.

When examined in December, the patient was very fit and well; there was no sign of any irregularity in the chest wall; the defect in the fourth rib could hardly be felt owing to new formation of bone under the periosteum.

The tumour after excision (*Fig. 106*) measured 7 in. by 6 in.; it was lobulated and of a pearly-blue colour. On section (*Fig. 107*) there were definite areas of ossification, and in one place where the radium had been inserted there was a mucoid degenerative area.



The microscopical picture (*Fig. 108*) was that of a typical chondroma. In the centre is seen a group of nutrient vessels, whilst the main feature of the tumour itself is that of apparently homogeneous cartilage containing large



*FIG. 108.* —Microscopical section of the tumour.

numbers of irregularly distributed but typical cartilage cells. The cells themselves vary greatly in size.

The specimen is now in the museum of the Royal College of Surgeons.

## RENAL COLIC AS A LATE COMPLICATION OF NEPHRECTOMY.

By H. W. B. CAIRNS, LONDON.

THE following case presented the unusual feature of a renal colic occurring eleven years after removal of the corresponding kidney. The cause of this colic and of the other symptoms, which were equally baffling, was discovered by mere chance.

**HISTORY.**—H. N., male, age 36, was admitted to the London Hospital on March 17, 1923. Ten days earlier he had developed suddenly a typical left-sided renal colic: the pain began in the left loin and radiated to the left lumbar region and the hypogastrium; it was severe and intermittent, and was accompanied by frequent and painful micturition.

The colic had continued, rendering sleep almost impossible, until twenty-four hours before admission, when it suddenly ceased. Three hours later the patient felt a sudden stoppage of the stream while trying to pass water. He strained hard, but could only pass a little blood, while at the same time he felt a stinging pain in the penis. Seven hours later he was still unable to pass water, so his doctor passed a catheter; the relief, however, was only temporary, for when he was admitted to hospital he had acute retention which required immediate catheterization.

PAST HISTORY.—It appeared that the patient had had urinary trouble before. In 1911 he had been in the London Hospital on account of left renal colic, and Mr. Frank Kidd had removed a large stone from the pelvic portion of the left ureter. The ureter, it is stated in the notes, was greatly dilated, "resembling the sigmoid colon". The incision in the ureter was closed by a double layer of chronic catgut stitches. A fortnight after the operation a large, tender swelling appeared in the left loin. This proved to be a hydro-nephrosis, for which first nephrostomy, and finally nephrectomy, were performed. These operations were successful, and the patient returned to his work and had no further trouble, save for an occasional ache in the left loin, until the sudden onset of his present illness.

PHYSICAL EXAMINATION.—On admission, the only physical signs of importance were the distended bladder and a tenderness in the left loin at the site of the old healed nephrectomy incision. The right kidney was palpable, but neither enlarged nor tender. The urine was alkaline and contained  $\frac{1}{2}$  volume of albumin and a deposit of pus and red cells. On cultivation it yielded, later, *B. proteus*.

On the following morning during a further examination of the patient a curious discovery was made. Projecting from the urethral orifice there was a small mass— $1.6 \times 0.6 \times 0.5$  cm.—the surface of which resembled twisted string. It was removed and sent to the Pathological Institute. Professor Turnbull reported as follows:—

"The specimen consisted of longitudinal and transverse sections of anisotropic rods surrounded by a granular structureless substance. The morphology and staining reactions of the rods were similar to those of a silk ligature in a control section."

Some further examinations were carried out. X-ray examination of the urinary tract revealed no abnormality. The bladder was found at cystoscopy to be normal; the ureteric orifices were of equal size.

SUBSEQUENT COURSE.—After the expulsion of the stitch the patient had no further symptoms. He was discharged from hospital on March 26, 1923, and has reported since that there has been no subsequent trouble.

### SUMMARY.

This case would have remained a mystery if we had not had the good fortune to discover the stitch. This made the course of events perfectly clear. At the nephrectomy operation the distal cut end of the dilated ureter was evidently closed by a silk ligature. A slight degree of infection persisted around this fragment of silk, causing from time to time a slight aching pain

in the left loin. Twelve years after it had been placed in position the silk worked loose and was expelled down the still patent ureter to the accompaniment of a sharp attack of renal colic. In the bladder it gave rise to a temporary retention of urine, which could be easily relieved by catheter. Finally, the ligature was discharged at the external meatus of the urethra.

The case emphasizes a point established in recent years, namely, that non-absorbent material should not be used in nephrectomy for ligature of the ureter, particularly when the ureter is dilated. The case also gives an excellent example of the *honesty* of symptoms; in it, as in so many other obscure cases, once the problem is solved, symptoms which have at first sight appeared complicated, incongruous, almost unbelievable, are now seen to be an eminently reasonable expression of the underlying pathological processes and of their development.

My thanks are due to Mr. H. S. Souttar for permission to publish this case.

## CYST-ADENOMA OF PANCREAS: COMPLETE REMOVAL.

By SETON PRINGLE, DUBLIN.

THE following case of enucleation of a cyst-adenoma of the pancreas seems to be worthy of record.

Mrs. D., age 40, consulted me in February, 1924, for a "lump in the left loin". She stated that it had been there for many years—probably ten—but at first caused no inconvenience. It gradually increased, however, until at the time of seeking advice the size was such as to interfere with her stooping or even sitting down, the act of bending forward apparently causing it to be nipped between the lower margin of the thorax and the iliac bone. At no time had she suffered any pain of severe character, but latterly there had been occasional vague pains in the left upper abdomen, quite distinct from the acute discomfort produced by stooping. For some months she had had slight frequency of micturition, with some loss of control. The tumour did not interfere with her general health, which had always been very good, apart from an attack of acute rheumatism fourteen years previously.

On examination, it was found that almost the entire left abdomen was occupied by a smooth rounded tumour, extending from the loin behind to almost the mid-line in front, and from underneath the left costal arch to within a hand's breadth of Poupart's ligament. The percussion note over it was dull, except where the lower and left lateral borders were crossed by a broad zone of resonance, due to the transverse and descending colon. The stomach was displaced upwards, lying entirely under the cover of the ribs. On account of the history of some disturbance of micturition, careful examination of the urine was made, but it proved to be quite normal. Cystoscopic examination revealed a normal bladder, but the excretion of indigo-carmin by the left kidney was slightly delayed.

My friend, Dr. Campbell Hall, of Monaghan, assisted me at the operation. On opening the abdomen we found that the tumour presented through the

thinned-out, stretched gastrocolic omentum. This was divided freely, opening into the lesser peritoneal sac; the tumour—as large as a football—was then seen, still covered with the posterior parietal peritoneum, but its size was such that it was impossible to establish its origin. It was obviously cystic in nature, and a large trocar was inserted and some four pints of light chocolate-coloured fluid were aspirated. It then became apparent that it was attached by a broad base to the posterior abdominal wall in the region of the pancreas. At first our intention was to marsupialize the cyst wall, but this was so extensive that we proceeded to excise the redundant portion. The edges of the incision in the cyst wall proper were caught in forceps, and an

attempt was made to peel off the covering layer of tissue with gauze-covered fingers, just as a hernial sac is dealt with. This was accomplished with such ease that the process was continued until the whole cystic mass was removed. There was no serious hæmorrhage, and only two ligatures were applied, as the large vessels which ramified over the tumour peeled off with the covering tissues. After removal it was seen that the last portion of the cyst to be

shelled out had left an area denuded of peritoneum some 2 in. square on the anterior aspect of the pancreas in its middle third, so that the gland tissue was left quite bare and oozing blood; but there did not seem to be any actual removal of the glandular tissue, and

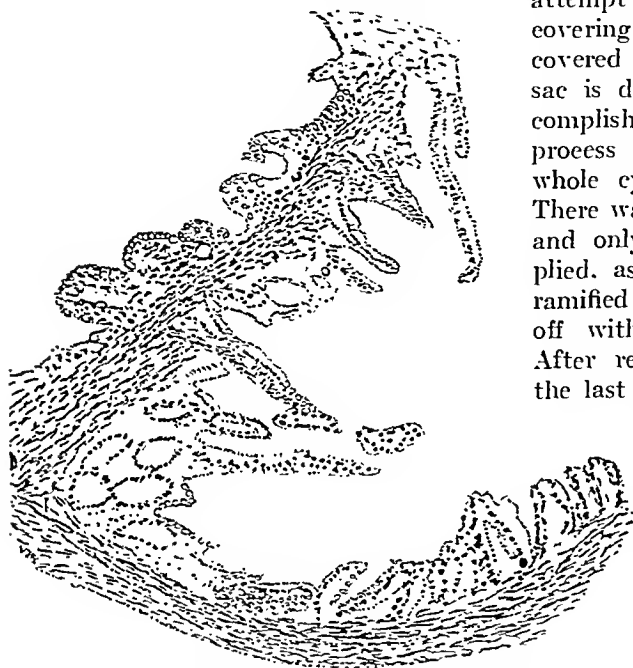


FIG. 109.—Sketch showing microscopic appearances of multilocular cyst-adenoma of the pancreas.

none could be identified with the naked eye as adhering to the excised cyst.

Having satisfied ourselves that no serious hæmorrhage was occurring, the cavity was loosely packed with gauze soaked in liquid paraffin, a large drainage tube inserted, and the edges of the opening were sutured to the anterior parietal peritoneum, thus shutting off the general peritoneal cavity.

Convalescence was uninterrupted except for a rather profuse venous hæmorrhage from the cavity in the second week.

The Pathological Department, Trinity College, Dublin, reports as follows:—

“When examined, the tumour was about 6 in. in diameter, a large quantity of the fluid having been drawn off or escaped. It was composed of one large cyst and a number of smaller ones in close association. The lining

membrane of these cysts was mostly smooth but sometimes velvety in appearance. The small cysts contained a gelatinous material.

"Microscopically, the cysts are lined with a single row of high columnar epithelium with the nuclei at the base, and showing many goblet cells. These are arranged in three forms: as a uniform row, as a singularly regular row of short papillæ, and as a row of long and occasionally branched papillæ; but in every case the single layer is maintained. The stained contents of the cysts are mucous threads and many desquamated cells. The wall dividing the cysts is formed of fibrous tissue, which is sometimes hyaline but more commonly cellular. There is a plentiful blood-supply and occasionally some hæmorrhage—this last possibly due to handling at and after the operation. There are no remains of pancreatic tissues in the walls of the cysts, such as the tissue resembling islands of Langerhans sometimes described in pancreatic cysts. The tumour on the whole closely resembles a cyst-adenoma of the ovary. It is apparently entirely benign, with no evidence of infiltration. (*Fig. 109.*)

"The contents of the main cyst were examined by Dr. Horgan, who reports that he has found all the pancreatic ferments.

"This tumour is to be distinguished from retention cysts of the pancreas by the formation of the papillæ, and by the comparative ease with which it was removed. It is to be distinguished from enteric cysts, which are sometimes found embedded in the mesentery, by its attachment to the pancreas, its multilocular formation, and the presence of pancreatic ferments."

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## ECTOPIC OVARY.

By ARTHUR EVANS AND STANFORD CADE, LONDON.

CASES of incompletely descended ovary are of sufficient rarity to be worthy of record.

Miss E., age 22, was sent to hospital with a diagnosis of recurrent attacks of appendicitis. Her health had been good up till eighteen months before admission. She then had periodical attacks of pain in the right side of the abdomen; these attacks were very severe, and the pain lasted for about twelve hours. There was no vomiting, only nausea occasionally. Pain and tenderness on pressure in the right iliac fossa usually persisted for a few days after each attack. The periods were normal, and had no particular relation to the pain.

The usual gridiron incision was made in the right iliac fossa; on opening the peritoneum the ovary presented in the wound. The cæum and appendix were normal. The ovary, normal in size, was situated on the outer side of the cæum. It was sessile, and partially covered by peritoneum. The uterus was mal-developed, and presented the appearance of a left uterus unicornis. The left ovary, tube, and broad ligament were normal. The right broad ligament was absent. The right Fallopian tube was represented by its fimbriated extremity. The ovary occupied a foetal position with cephalic and caudal poles. From the caudal pole a band stretched downwards towards the brim of the pelvis (ligament of ovary), where it bifurcated, one part going towards

the internal abdominal ring (round ligament), the other band stretching down to the uterus and representing the ill-developed right cornu. In order to remove the ovary the peritoneum attaching it to the posterior abdominal wall

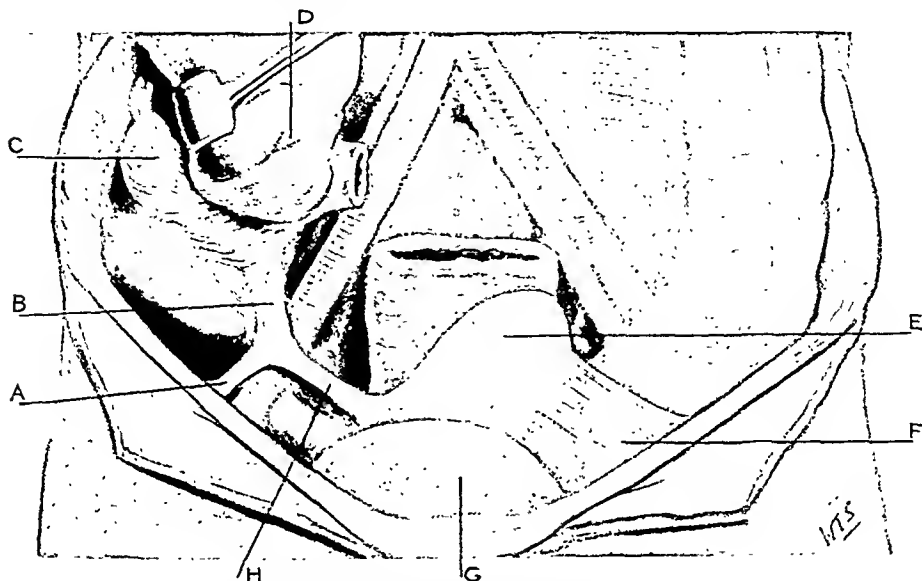


FIG. 110.—General position of pelvic viscera as seen at operation. A, Right round ligament; B, Ligament of right ovary; C, Right ovary; D, Cæcum; E, Left cornu of uterus; F, Left broad ligament; G, Bladder; H, Right cornu of uterus.

had to be excised with it. The patient had an uneventful convalescence. The attacks of pain did not recur.

In the normal course of events the ovary develops in the lower dorsal region. It possesses a mesovarium, and lies close to the Fallopian tube and mesosalpinx; the mesovarium and mesosalpinx are attached to the posterior abdominal wall by a common genital mesentery. The ovary reaches the iliac fossa in the fifth month of gestation, the brim of the pelvis at full term, and its normal position in the 'ovarian triangle' after birth. During its descent the mesovarium, the mesosalpinx, and the common genital mesentery join to form the broad ligament. It is obvious that in the case of non-descent the broad ligament cannot be formed. This was well illustrated in the case described.

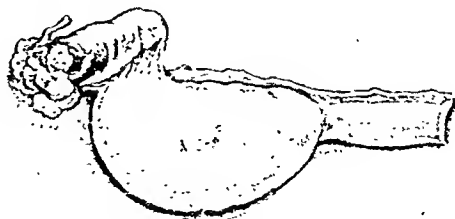


FIG. 111.—Ectopic ovary.

Fig. 110 shows the general position of the pelvic viscera as observed at operation. Fig. 111 represents the ovary as it appeared after removal. Microscopical sections of the cut end of the specimen show unstriped muscle, indistinguishable from that found in the normal uterus.

## A CASE OF HEART SUTURE.

By HAMILTON DRUMMOND, NEWCASTLE-UPON-TYNE.

J. W. S., age 25, a sailor, was admitted to the Royal Victoria Infirmary at 7 p.m. on July 8, 1924, with the following history: Owing to a disappointment in a love affair, on the day of admission at 6 p.m. he attempted to take his life by stabbing himself in the heart with a penknife. He immediately collapsed in the street, and was shortly afterwards seen by Dr. Eric Dagger, who found him lying on the pavement drunk and with a considerable amount of blood on his clothing. He was immediately despatched in an ambulance to the Infirmary, a distance of about two miles.

On admission he looked pallid and was under the influence of alcohol; his general condition was fairly good, pulse 80 and regular, temperature

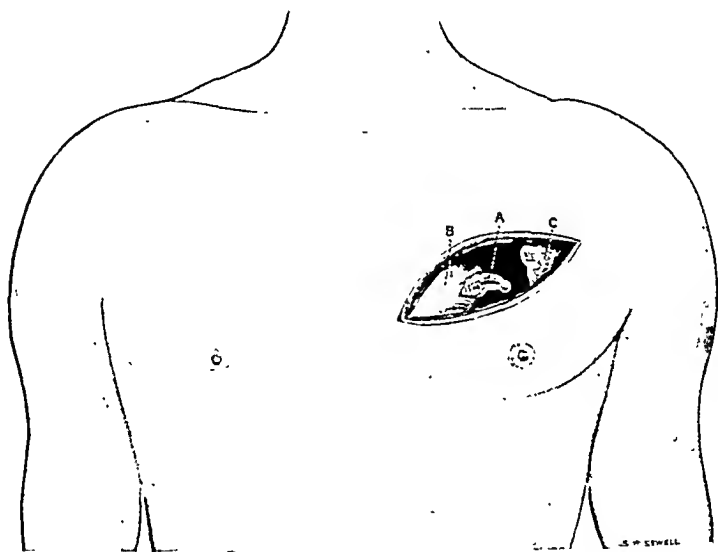


FIG. 112.—Shows condition found at operation and the incision employed—excision of third rib cartilage with portion of adjacent rib. A, Portion of left auricle protruding through pericardium; B, Pericardium; C, Lung.

normal. His respirations were not increased, nor had he any dyspnoea. There was a small stab wound situated in the second intercostal space on the left side,  $\frac{3}{4}$  in. long and  $1\frac{1}{2}$  in. from the sternum. There was not any hæmorrhage from the wound, and it admitted a probe for about 1 in., the pectoral muscle being well developed. There was no increased cardiac dullness suggestive of pericardial effusion.

On account of the absence of grave symptoms the wound was dressed, and the likelihood of any serious injury was not entertained. About an hour later the patient became restless and complained of difficulty in breathing.

He was more pallid, and preferred to be in a half-sitting posture on his right side. Dyspnoea became rapidly worse, respiration was rapid and shallow, and his pulse rose to 110. He was then seen by Dr. Horsley Drummond, who found dullness in the left chest up to the level of the sixth rib; there was still no increased dullness of the pericardium, but the heart was displaced to the right. Immediate operation was advised, as it was now clear that he was suffering from a progressive hæmorrhage into the left pleural cavity.



FIG. 113.—Shows line of the incision, taken ten weeks after the injury, shortly before leaving hospital.

It was impossible to carry out an operation with a local anæsthetic because he was so restless and continually throwing himself about in his attempts to breathe more freely. Open ether was given, and the wound explored by an incision 6 in. in length along the third rib cartilage, splitting the pectoral muscle and extending along the third rib (*Figs. 112 and 113*). The costal cartilage and one inch of the rib were excised. The left pleura was freely opened for the length of the incision, the anterior margin of the left lung was pulled up, and the lung examined; it was uninjured. Deeper inspection showed, at the base of the pericardium on the left side, a hole  $\frac{1}{2}$  in. long through which the left auricular appendix protruded, and from it there



was a continuous flow of blood. The wound in the auricle was practically at its tip (*Fig. 114*), and on catching the edges with fine dissecting forceps a rush of blood escaped from the heart.

The auricular wound was closed with catgut sutures; but this was a difficult procedure owing to the thinness of the wall of the auricular appendix, which at the point of injury did not appear to be thicker than a child's omentum. After three interrupted catgut sutures were introduced the hæmorrhage was controlled. Another difficulty in the closure of the auricle was caused by the continual welling-up of blood from the left pleural cavity. The major part of the blood in the pleura was mopped out—probably 60 oz.—and the pleural cavity was closed completely without drain by a row of interrupted catgut sutures for the pleura and a second layer for the pectoral muscles.

The patient greatly improved in condition after the operation, and was no longer restless. On the following day his general condition was fair; respirations were rapid—68, largely due to a pneumothorax; temperature 100°, pulse 116.

On July 11 he was much improved, respirations 56, temperature 99.2°, and pulse 108, though there was a good deal of expectoration, and signs of considerable pleural effusion were present. The following day he was about the same: the pleural effusion appeared to be diminishing. Steady improvement occurred until the 22nd, fourteen days after operation, when his temperature rose and dyspnœa was more marked.

Twelve ounces of blood-stained serum were removed from the pleural cavity by aspiration, and on bacteriological examination it proved to be sterile. For several days he complained of severe precordial pain, and this was accompanied by vomiting. A considerable amount of consolidation of the left lung was present at the base, and there was rusty sputum.

An empyema of the left chest developed, which was aspirated on Aug. 19, and operated upon under a local anæsthetic. A small portion of rib was resected and the chest drained. The pus on examination showed pneumococci and staphylococci. After this his recovery was uninterrupted, and he left hospital healed and well on Sept. 29.

Since leaving hospital, patient has been seen from time to time and is in excellent health. (February, 1925.)

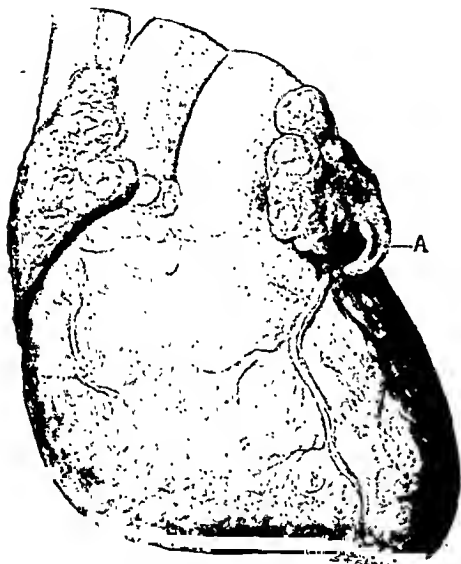


FIG. 114.—Drawing of a normal heart.  
A shows position of the wound in left auricle.

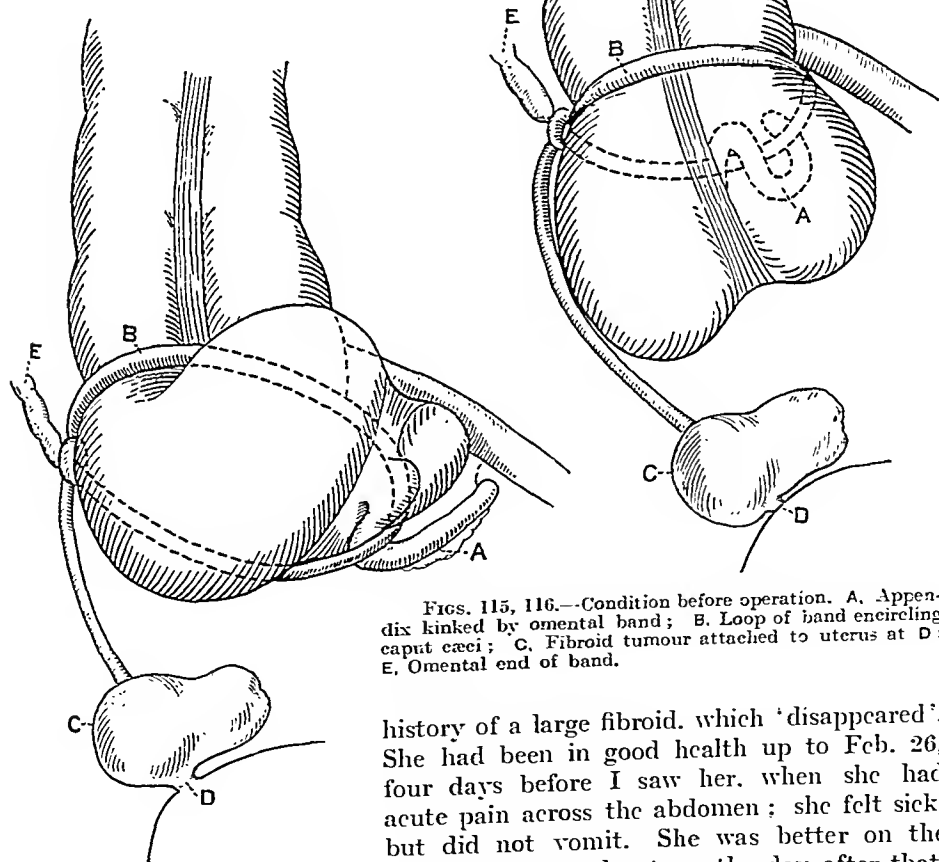
## CASE OF SNARING OF THE CAPUT CÆCI AND APPENDIX BY AN OMENTAL BAND.

By SIR HUGH M. RIGBY, K.C.V.O., LONDON.

SURGERY of the abdomen owes much of its interest to those unexpected and almost dramatic findings which are occasionally met with in the course of one's routine work. The following recorded case is an example :—

On March 2, 1925, I was asked to see in consultation a lady with the following history :—

The patient had had one child, two years previously ; labour was prolonged, and the child still-born. There was a



FIGS. 115, 116.—Condition before operation. A, Appendix kinked by omental band; B, Loop of band encircling caput cæci; C, Fibroid tumour attached to uterus at D; E, Omental end of band.

history of a large fibroid, which 'disappeared'. She had been in good health up to Feb. 26, four days before I saw her, when she had acute pain across the abdomen ; she felt sick, but did not vomit. She was better on the following day, and got up the day after that,

though she felt pain in the right side. Three days after the onset of the attack, she was seen by her doctor : there was then no rise of temperature ;

the pulse-rate was 70. Local tenderness was noted in the abdomen, and some rigidity of the muscles; pelvic examination was negative. The monthly period coincided with the attack, and the significance of this was considered. Discomfort was still present. When I saw her, the temperature was normal and the pulse quiet. On pressure over the ileocaecal region, distinct tenderness was felt, and some pain was also noted on pressure to the left of the mid-line. A diagnosis of appendicitis was made and operation was advised.

OPERATION, March 6, 1925.—Under anaesthesia, an ill-defined lump could be felt in the right iliac region.

The abdomen was opened by a right paramedian incision. The caecum was at once exposed to view, and the following very unusual condition was disclosed (*Figs. 115 and 116*).

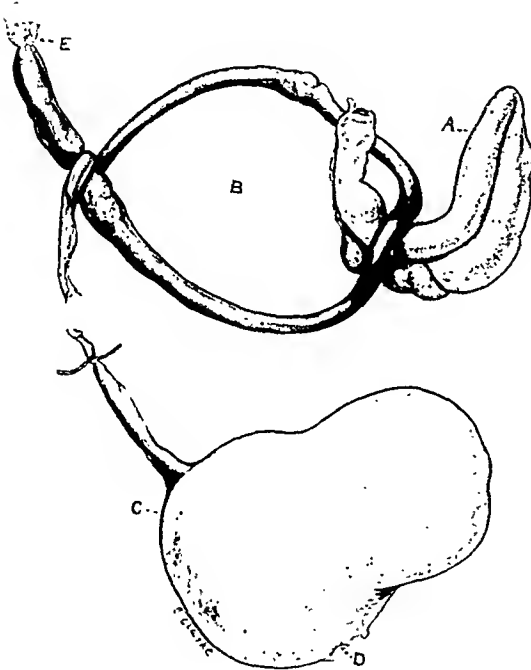


FIG. 117.—Specimen removed at operation and replaced in position. A, Appendix kinked by omental band; B, Loop of band encircling caput caeci; C, Fibroid tumour attached to uterus at D; E, Omental end of band.

Surrounding the caput caeci was seen a string-like band of deep maroon colour, adherent to the caecal wall at the outer side; here, a knot could be seen, and, from this knot, the band passed downwards over the brim of the true pelvis. The caecum was somewhat mobile; on lifting it up, the band was seen to pass across the appendix, near its attachment. At the point of crossing, a deeply injected groove was seen, so that the appendix was acutely kinked; the distal part was a little distended, but of good colour, and the mesentery apparently was not congested. On tracing the band downwards, it was found to be attached to a smooth tumour in the right side of the pelvis, which at first was thought to be an ovarian cyst. The band was ligatured at the pelvic brim and divided; the knot was then untwisted, and the part of the cord encircling the caput caeci was

freed; in parts it was adherent, but in the main it could be separated without difficulty. The band was then traced upwards, and was found to be continuous with the right free margin of the great omentum. The upper end was ligatured and cut across. The appendix was then removed, and the caecum and lower part of the small gut returned to the abdominal cavity.

The pelvic tumour was next examined: it proved to be a subserous, pedunculated fibroid attached to the right side of the fundus of the uterus by a short pedicle; the latter was ligatured and the tumour delivered from the pelvis. The caput caeci was not engorged, and, although fairly tightly

constricted by the band. showed no appreciable damage to its coats at the site of the constriction. The abdominal wound was then closed.

Immediately after the operation, a drawing was made, the parts removed being reproduced as shown in *Fig. 117*; the loop made by the band is just as it was found, and the appendix and tumour are replaced in their original position. My secretary, Miss Ethelwyn Clarke, has made an exact picture.

The knot formed by the band was produced by a double twist of the loop on its axis. Presumably the band was formed at or after the pregnancy occurring two years previously, but how the caput caeci became involved, and how the double twist occurred, must remain a matter of speculation.

## TWO CASES OF FOREIGN BODIES IN THE GASTRO-INTESTINAL TRACT.

By WILMOT ADAMS, PENANG.

### 1. Cast of Vegetable Material Simulating Carcinoma of the Stomach.—

A Chinese male, age 37, was admitted under my care on Nov. 5, 1921, complaining of persistent vomiting, loss of weight, and gastric discomfort. These symptoms had been present for about five months. No history of hæmatemesis or melæna. He was markedly wasted, but otherwise his general condition was fairly good. In the epigastric region was a hard tumour which corresponded roughly to the shape of the stomach. No peristalsis was elicited, there was no free fluid in the peritoneal cavity, and the liver was not enlarged.

I considered it to be an advanced case of carcinoma of the stomach, and that on account of its extent even a palliative operation was out of the question. This was explained to the patient, who begged me to explore in order to make perfectly certain. This was done on Nov. 8, under local anæsthesia. Local infiltration of the abdominal wall with  $\frac{1}{2}$  per cent novocain was the method employed.

On opening the abdomen and palpating the stomach, it immediately became obvious that the tumour lay within the stomach, the walls of which moved freely over it. The stomach was delivered and packed off, and an incision 5 in. long made in the long axis midway between the two curvatures. The foreign body was removed, and proved to be a cast of the stomach composed of some vegetable material. The pylorus was patent, and there was no sign of ulcer or neoplasm. The incision in the stomach was closed in two layers in the usual way, the packing removed, and the abdominal wound sutured in layers.

On examination, the cast was found to be composed of pumpkin. There was no macroscopic evidence of any other foreign body forming a nucleus. Recovery was uneventful.

### 2. Richter's Hernia-scrotal Abscess Containing Fruit Stones.—

A Chinese male, age 60, was transferred to my care from the Pauper Hospital on July 7, 1923, for operation for strangulated inguinal hernia. He stated he had had the hernia for eight years, but that during the last two months it had been irreducible and painful. The hernia appeared to be a typical strangulated inguinal one. The patient's general condition was excellent.

There was no abdominal distention or other evidence of general peritonitis or mechanical obstruction. He was quite positive that his bowels had moved that morning, and he had only come to hospital on account of the pain.

Under local anaesthesia (infiltration with  $\frac{1}{2}$  per cent novocain) herniotomy was performed at once. The sac contained a loop of small intestine, the apex of which was firmly adherent to the lower end thereof. In endeavouring to separate it I opened into an abscess containing about 4 oz. of foul-smelling pus. On exploring the abscess cavity, I found it contained three hard foreign bodies, which on opening up further proved to be three rambutan stones. They are about the size of plum stones. Investigating further I found that the gut was perforated, and in such a way had compression been exerted that the portion below the point of strangulation (the external ring) had been converted into a funnel-shaped process, the wide end of which opened into the abscess cavity, but the gut was not completely occluded. This portion was markedly hypertrophied. The mucosa was thick and rugose, and the rest of the wall almost fibrous in consistency.

It was obviously essential to remove this portion, and it was clear that after doing so the lumen of the gut would be too narrow for safety if simply local removal was performed. I therefore resected 8 in. of gut, including the strangulated portion, and made an end-to-end anastomosis with clamps in the usual manner. The gut was returned, and the sac cleared up to the internal ring and removed, but the neck of the sac was not tied off. Two catgut sutures were passed through the neck of the sac and made to interlock, but were left untied. A short rubber tube was passed through the internal ring down to the site of the anastomosis. The skin wound was loosely sutured, and a tube put in at the lower end.

The patient was kept on dilute brandy and water by mouth, and rectal salines, for four days. The bowels moved naturally after thirty-six hours. An enema was given on the fourth day with a good result. On the fifth day, no leak from the anastomosis having occurred, the tube in the internal ring was removed and the neck of the sac tied off. Subsequent recovery was uneventful, except for inevitable suppuration in the wound, which took five weeks to heal. On account of this, and the age of the patient, it did not appear advisable to attempt anything further in the nature of a radical cure. He was instructed to report in three months' time, or if any swelling reappeared before that date.

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## SARCOMA OF THE RENAL PELVIS, ASSOCIATED WITH HYDRONEPHROSIS.

By F. G. RALPHS, DUKINFIELD.

A WOMAN, age 39, was referred to the Ashton-under-Lyne Infirmary with the diagnosis 'twisted pedicle of ovarian cyst'. On admission, her general condition was one of collapse, with a small pulse of 130 and a subnormal temperature. Two days previously she had first noticed a swelling on the right side of the abdomen.

ON EXAMINATION.—A circumscribed mass was revealed to the right of the umbilicus, and continuous above with a large elastic tumour filling the right hypochondriac and lumbar regions. The tumour showed slight lateral mobility, and was not tender to palpation; a thrill could be elicited across its lower border.

Prior to the appearance of the swelling, she had experienced no discomfort save an occasional aching pain in the right loin. There was no history of hæmaturia.

OPERATION.—Vaginal examination having excluded the pelvis from involvement, laparotomy through a right paramedian incision was performed. A large bluish and cystic tumour presented, bulging forward the posterior peritoneum and occupying a large extent of the retroperitoneal space on the right side. The cæcum and ascending colon were adherent to the medial aspect of the tumour; the appendix stretched transversely across its lower pole. The texture of the tumour varied appreciably, for whereas the lower portion was cystic and elastic to the touch, the upper portion was reniform and of firm consistence. The whole mass, enclosed in a thick fibrous capsule, appeared to be a large hydro-nephrotic kidney with a solid growth at the upper pole. Separation of the proximal colon by gauze stripping, and free incision

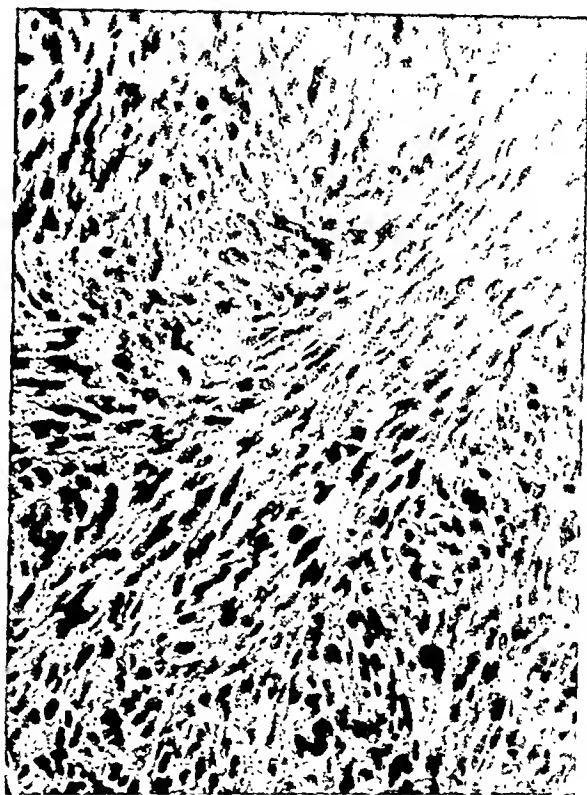


FIG. 118.—High-power view, showing spindle cells.

of the peritoneum and fibrous capsule, permitted of enucleation of the tumour from its retroperitoneal bed. There was free oozing at this stage, controlled by a big gauze pack. Access to the renal pedicle was barred by the formidable size of the mass, which was diminished by tapping the cystic lower portion. About one pint of blood and clot escaped, and facilitated exposure of the pedicle. This was thick and adherent to the vena cava. During the separation of these adhesions the patient became very collapsed, and therefore nephrectomy was rapidly completed. The tumour, on removal, weighed 28½ oz.

Of necessity, the after-history is brief, for the patient died a few minutes after the operation. Her pulse had failed during the isolation and separation of the renal pedicle from the vena cava. Some degree of traction was unavoidable, and doubtless contributed to the fatal result.

**PATHOLOGICAL FINDINGS.**—To the naked eye, the tumour presented two strongly contrasting parts—an upper, of soft, diffuent, brain-like tissue encased in a stout fibrous capsule, and a lower, composed of a large hydronephrotic sac, into which a copious hæmorrhage had occurred. It is probable that the development of the hydronephrosis was secondary to a gradual obstruction of the renal pelvis by a growth therein, for such was the origin of the growth removed, according to the pathological report appended. I am indebted for it to the kindness of Dr. Powell White, of the Cancer Research Laboratories of the University of Manchester.

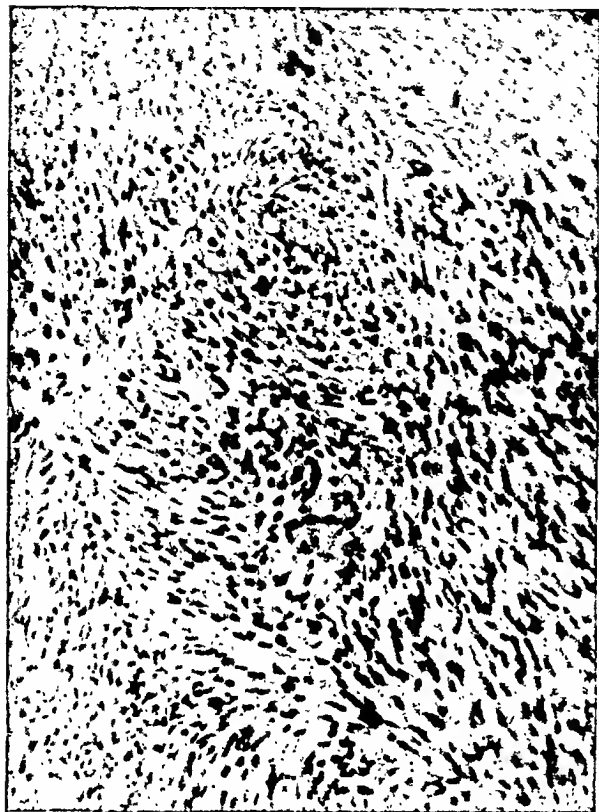


FIG. 119.—Low-power view through solid portion of growth.

**PATHOLOGICAL REPORT.**  
—“The kidney tumour is a spindle-celled sarcoma. It shows some mucoid degeneration in places, and a considerable amount of necrosis and hæmorrhage. I should

think it originated in the connective tissue of the renal pelvis. I was able to find some small remains of kidney tissue.”

**OBSERVATIONS.**—Malignant tumour of the renal pelvis is an uncommon type of growth, to judge from the rarity of its occurrence in surgical literature. In this example, there seems to have been a striking absence of symptoms until the stage of degeneration and necrosis had been reached.

## REVIEWS AND NOTICES OF BOOKS.

*Papers and Addresses in Surgery (Selected and Revised).* By R. HAMILTON RUSSELL, F.R.C.S., Associate of King's College London: Consulting Surgeon to the Alfred Hospital, the Children's Hospital, the Victorian Eye and Ear Hospital, and the Queen Victoria Hospital for Women and Children, Melbourne. Demy 8vo. Pp. 452, illustrated. 1923. Melbourne: Allan Grant.

SURGEONS who have been interested in the writings of R. Hamilton Russell—and they exist all over the world—as well as his friends and former students, will be glad to have his more important papers collected into one volume. In the preface the author states that the publication would never have seen the light of day but for the knowledge that the task was about to be undertaken by others. We can be grateful that this induced him to edit the papers himself.

Mr. Russell is a surgeon who has never been satisfied to follow text-book procedures unquestioningly. Every case has come to him as a problem in principles. Very early in his practice at the Children's Hospital, Melbourne (*Lancet*, 1899) there appeared to him to be flaws in the accepted etiology and pathology of indirect inguinal hernia. This doubt resulted in his belief in a congenital sac as the sole cause of indirect inguinal hernia. Investigations in the post-mortem room (p. 123) lent colour to this theory to the extent that a sac was found to be present in a high percentage of individuals who had not been the subject of hernia. The only proof of the theory which seemed possible was to put it to the operative test—to remove the sac at its origin, not near its origin—and to omit all procedures usually considered necessary for strengthening the abdominal wall. His theory has been put to this severe test in that for many years he, his colleagues, and successors have abandoned any other procedure beyond high ligation of the sac in this type of hernia, except where advancing years or the size of the hernia had weakened the anatomical structures surrounding the neck of the sac.

Mr. Russell's theory of the causation of properitoneal and interstitial hernia will be read again with interest. Everyone does not yet agree with him as regards the sacular origin of femoral hernia, but all surgeons will, after reading the chapter, be alert to the possibilities, and on the look-out for evidence. The reviewer had the good fortune to see recently a large femoral hernia in which the sac descended between the femoral artery and vein—apparently a congenital sac caught up by the growing limb bud in foetal life, as Mr. Russell suggests. There will also be differences of opinion as regards the operative procedure to be followed in femoral hernia. The high operation is being performed more and more, and, as far as can be judged from the follow-up department in one hospital, with far fewer recurrences.

The chapters on fractures—simple and those implicating joints—are eminently sane, and his scathing criticism of passive motion is well merited; the simplicity of the methods advocated, and the unremitting personal attention of the surgeon, are two points well worthy of attention.

Intractable stricture of the urethra often drives surgeon and patient to despair. Mr. Russell's idea of dealing with the very old man (p. 295) by "deliberately converting the subject of intractable stricture into a case of hypospadias", with its happy result, was an inspiration. Treatment of difficult strictures by excision has been practised, but hitherto the urethra has been completed by accurate suturing. The results were such that the methods had fallen into disuse. Realizing that if the urethra be slit up ventrally for any length and left open, perfect repair and function will result, Mr. Russell has devised an operation by which the urethra is slit up in this fashion, making the tube of mucous membrane into a ribbon. The



stricture is excised, and the ends of the ribbon are united by stitches and left as a ribbon, no attempt being made to convert it into a tube. The absolutely free drainage ensures perfect healing of the mucous membrane; the tube reforms itself as the wound heals, and, as the exciting cause and the cicatricial tissue have both gone, the urethra remains patent and soft. Details of the operation, with many illustrative cases and the late results, are given.

Two articles are noteworthy in that procedures which at the time were unique have in later years become common practice. In 1903 Mr. Russell freely opened a chest under general anaesthesia in order to remove a large-headed pin from a bronchus; and in 1907, when operating upon a patient very ill with an infected hydatid cyst of the liver, he evacuated the cyst and its contents, cleansed the cavity, filled it with saline solution, accurately sutured it, and then completely closed the abdomen. Nowadays pleural cavities are opened with impunity, and surgeons have learned that the abdominal cavity can deal with some types of infected material with safety; but at that period both procedures required vision and courage.

As a teacher one realizes that Mr. Russell loved his students, and, by his own life and attitude towards his profession, ever kept before them a very high ideal of the calling they had chosen. The chapter with which the volume opens, the Presidential address to the Section of Surgery, Australasian Medical Congress, Auckland, N.Z., in February, 1924, is charged with reverence and affection for Lord Lister, Mr. Russell's chief when a student and house surgeon at King's College Hospital; and, just as much of Lister's mind and practice live in Mr. Russell, so are the best traditions passed on again by Mr. Russell to the next generation. The surgeon who compels thought, and whom his students and associates love, achieves greatly more than the acceptance or non-acceptance of his own theories.

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**Surgical Operations: A Text-book for Nurses.** By E. W. HEY GROVES, M.D., B.Sc., M.S., F.R.C.S., Surgeon Bristol General Hospital, Professor of Surgery, Bristol University. Second edition. Royal 8vo. Pp. 255, illustrated. 1925. London: Humphrey Milford, Oxford Medical Publications. 21s. net.

THAT this unique work was long overdue all who read the first edition must have felt, and that it has come to stay is happily assured by the arrival of a second edition. Whether approval is universal or no, among those who care for the sick, the modern probationer with an average secondary school education will and should no longer be satisfied with 'blind' nursing. As in others, so in her profession, science is invading, and the nurse blends tireless devotion to her patient and the doctor's instructions with a desire to know 'what was done in the theatre' and 'why'. Thus she gains an intelligent interest in her work, and becomes a more harmonious medium between the surgeon and patient. Her cardinal virtue of constant loving care can only be enhanced by an understanding mind.

To represent the vast range of modern operations in a brief, simple, and picturesque form is a bold venture, but the reputation of the author of *Surgical Operations* is commensurate with it. There is an admirable selection of operations, and the descriptive paragraphs contain the 'pith of the matter', whilst the abundant illustrations make a feature that cannot be over-praised. It is a pioneer work handled in a masterly fashion, and it has secured a prominent place in surgical literature which a few friendly criticisms will not impair.

In the opening chapter the text would be helped by illustrations of a modern operating theatre with operation proceeding, of a surgeon attired, and an operating table. The Trendelenburg position might well be figured. With nurses as readers the notes on the pre- and post-operative care of cases are specially welcome. They might be amplified in the chapters on tongue and pelvic operations, and the need for an empty bladder in pelvic cases deserves a note. One looks in vain for a reference to perineorrhaphy.

The illustration of instruments is a distinctive boon to the student, but space could be saved by not depicting many of them twice, i.e., in the text and in the appendix, which would then be available for some common examples at present omitted, e.g., proctoscope, various scissors, mouth gags, aural specula, etc. The

alphabetical arrangement of instruments seems no advantage over the common commercial custom of listing them in regional sets. With the increased delicacy and complexity of instruments it would be as well to add a note of warning to those in charge of them, indicating that it is not enough to assemble them only, but that their constituent parts must fit and their efficiency be tested before they are offered to the surgeon (and the patient!) for use.

These are a few trifling corrigenda, and one slip that certainly calls for remark is the description of the myxœdematous patient as having "greasy and perspiring skin".

In concluding one's remarks on *Surgical Operations*, one may say no nurse should be without it, and the student will warmly welcome so simple and comprehensive a portrayal of this fascinating subject when he embarks on the study of surgery.

**Surgical Pathology.** By JOSEPH MCFARLAND, M.D., Sc.D., Professor of Pathology in the Medical Department of the University of Pennsylvania. Royal 8vo. Pp. 701 + xiv, with 435 illustrations. 1924. London: H. K. Lewis & Co. Ltd. Price £2 2s.

The outlook of the author is epitomized in the first sentence of the preface, in which he tells us that "surgical pathology is not a separate subject, but merely the bringing together and emphasizing of those aspects of pathology that have been found to be of particular interest and importance to surgeons". The attempt to attain this object has resulted in this book, in which a great deal of useful information is to be found. Nevertheless, it is often disappointing, and much that would be of great interest and value to surgeons is not to be found in its pages.

The work is divided into three sections: (1) Congenital conditions of surgical interest; (2) Tumours; (3) Special pathology. Instead of starting with any general description of the pathological processes, that knowledge is assumed, and the author plunges directly into a detailed consideration of congenital conditions. This forms an interesting section of 202 pages which have been allotted from a total of 656. The classification adopted is very practical and useful, and the table of contents is in itself a synopsis of all the congenital anomalies which are ever likely to be met with. A good subsection is that on the persistence of the omphalomesenteric duct—Meckel's diverticulum—and it is well illustrated by diagrammatic representations of practically all the conditions found. Deformities like club-foot, club-hand, torticollis, congenital dislocation of the hip, etc., are also dealt with, but the author has nothing new or suggestive to say about them.

In Section II, devoted to the consideration of tumours, the author makes some pertinent remarks on the microscopic examination of tumours, and the relationship and mutual responsibility of the surgeon and of the pathologist in these matters. The classification of tumours is always difficult, and we are told that in the present uncertain state of our knowledge no classification can be regarded as satisfactory, but that the best is that of Adam, which is adopted in this work.

In dealing with the fibromas, we are reminded that the microscope is not an infallible guide in determining the malignancy or benignancy (*sic*) of tumours of this class. This is a point frequently emphasized in connection with many of the tumours, and all practical surgeons must find themselves in agreement.

Angiomas which give rise to secondary deposits in the viscera are described and figured. Myelomas are placed among the sarcomas, and the author also speaks of pseudo-sarcoma gigantocellulare in this connection. Altogether the paragraphs devoted to this subject do not tend to clarify the difficulties surrounding this type of new growth. Dealing with cysts, the author recognizes the difficulty of classification, and naively remarks that "the following tabulation seems to afford a convenient approach to the subject".

Section III deals with special pathology, or as we would say, regional pathology. The breast is very fully dealt with, and Bloodgood is freely quoted. Many technical points are considered as they would be in a text-book on surgery.

For those who have devoted much time to the study of naked-eye pathology, and who have insisted on its importance, it is refreshing to read, "Cancer is so

distinctive in its appearance that it ought to be recognized by the naked eye without difficulty". And again, "Cancer was never found by microscopic examination where it was not suspected from what the finger and naked eye had found, though occasionally what the eye and finger thought to be cancer was shown by the microscope to be something else". After these statements it is not surprising to find that the author does not place much reliance on the frozen section for diagnosis. A good deal of space is given to the consideration of sarcoma of the breast, and many clinical details are incorporated. Dealing with Paget's disease of the nipple, McFarland joins issue with Sampson Handley and considers that it is primarily a skin affection.

In the urogenital section, enlargement of the prostate receives much attention, and again many clinical points are dealt with. The author favours an inflammatory origin. In discussing ascending ureteral infection, no mention is made of Bond's careful researches on the ascending mucous currents, nor is this matter referred to elsewhere. The statement that sarcoma of the prostate is not recognized in adults is not in accordance with the recorded facts. In dealing with vesical papilloma, the author properly emphasizes the difficulty of deciding on its malignant nature or otherwise from a microscopic examination of a small portion—oftener obtained from the summit of the growth than from its base.

The gastric section is disappointing, and the question of the relationship of chronic ulceration to cancer is apparently evaded. In dealing with the pancreatic affections, inflammatory effusions into the lesser sac are not mentioned, and evidently the classical paper of Jordan Lloyd has been overlooked. The author has nothing new to say about the always interesting problem of intussusception, and the not infrequent inversion of Meckel's diverticulum is not mentioned.

The book concludes with a few pages devoted to the bones and joints.

There are 435 illustrations, most of which have been culled from other works. On the whole they are well selected, and have often been improved by being redrawn. It is pleasing to note that in every case the source of the borrowed illustrations has been properly acknowledged. There is also a useful bibliographic index, but a few inexcusable mistakes are obvious—Kelley for H. A. Kelly, Sill for Still, Shattruck, rendered elsewhere Shattuck, for Shattock, etc. There are also a good few misprints, and many peculiarities in spelling; some of these may only be Americanisms in common use, but they are not pretty words, and never likely to be acceptable to readers on this side.

This publication is not a text-book for students, nor will it replace any of the few existing works on the subject; nevertheless it contains much that is of interest to the practical surgeon, and should certainly be available for reference.

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**On the Breast.** By DUNCAN FITZWILLIAMS, C.M.G., M.D., F.R.C.S., Surgeon in Charge of Out-patients and Lecturer on Operative Surgery to St. Mary's Hospital. Royal 8vo. Pp. 440 xxv, with 166 illustrations and two plates. 1924. London: Wm. Heinemann. 30s. net.

THIS book is one of great practical importance: it contains a careful first-hand clinical account of the diseases and abnormalities of the breast. Its most valuable feature is the actual case history given of each typical condition. It would be easy to find fault with the book, but difficult to produce a better one. The description of microscopical anatomy, pathological theories, and the less direct methods of treatment such as radium is inferior to the clinical section of the work.

Mr. Fitzwilliams strongly opposes Handley's permeation theory, but his criticisms are based on general considerations rather than upon a study of morbid histology. He accepts the work of Lenthal Cheate in its entirety, without giving any very clear explanation of its purport.

The numerous illustrations are rather crude and diagrammatic, a large proportion being borrowed from other sources.

The outstanding merit of the book is its clear account of the clinical features of various diseases, and for this purpose we think it will establish a place as a *book of reference*.

**Dislocations and Joint-fractures.** By FREDERIC J. COTTON, A.M., M.D., F.A.C.S., Surgeon to the Boston City Hospital; Associate in Surgery, Harvard Medical School. Second edition. Pp. 745, with 1393 illustrations. 1924. London and Philadelphia: W. B. Saunders Co. 50s. net.

THIS second edition of a work which received full recognition on its first appearance is of great value. It has increased in size, it includes experience gained in the war, and it represents five further years of experience. Its value consists in the fact that it represents the personal observation and experience of one who has made very careful records. The illustrations are for the most part personal sketches or drawings by the author; and these, too, serve to keep the reader in actual touch with practical facts as they were observed at the time. For the most part the book consists in a minute description of all varieties of injuries near the joints, together with the symptoms and treatment. Details of treatment by manipulation or by splinting are very full, whilst details of operative treatment are conspicuously absent.

It will always form a valuable book of reference, although it will often have to be supplemented by a further work on operative surgery. For example, many pages are given to the description of the fractures of the lower end of the humerus; but one looks in vain for guidance in the treatment of a T fracture into the elbow when non-operative methods have failed. Similarly, the discussion of dislocation of the hip is given in great detail up to the point when manipulation has failed to reduce, and then we are left without guidance or advice.

**Lumbar Puncture: its Anatomical and Physiological Relations, Technique, etc.** With an Appendix on Encephalography and Puncture of the Cistern. By MARTIN PAPPELHEIM, M.D., Professor at the University of Vienna. Translated by George Caffrey. Denby 8vo. Pp. 248, illustrated. 1925. London: Bale, Sons & Danielsson Ltd. 15s. net.

THIS is a translation of the 1922 German edition, with a new appendix on encephalography and cistern puncture, and the addition of a few paragraphs on recent work. The majority of the book is taken up with details of the technique of lumbar puncture and of the examination of the cerebrospinal fluid, but thirty pages are devoted to some points of diagnostic value, and thirteen to "the therapeutical applications of lumbar puncture", chiefly in the treatment of syphilitic nervous diseases.

The arrangement of the book follows a plan common in German medical literature but less favoured in this country, viz., a complete absence of all sub-headings and italics, so that we often pass from one subject to another without noticing it. This is especially inconvenient in the chapters on technique, as the laboratory worker will have difficulty in finding any particular technical point in which he is interested, and the clinician will scarcely be tempted to sift the abundant chaff of laboratory detail for the few grains of clinical application which may be found among it.

In the details of the operation of lumbar puncture the clinician will find much that is useful, and also much with which he will disagree, as for example the recommendation that the needle should be made of steel, nickel-plated. "If a needle should break, it is usually easy to extract it with forceps; sometimes a skin incision is necessary."

The chapter on the diagnostic import of lumbar puncture will be of greater interest; as much space is devoted in it to the distinction between different forms of meningitis, *purulenta infectiosa*, *purulenta aseptica*, *concomitans*, *serosa*, and *meningismus*. It is not easy to obtain from it a clear perception of the meaning of these terms, and some confusion is introduced by such statements as that "*meningitis serosa* is characterized by clear, faintly turbid spinal fluid". The author's opinion that "in general the prognosis is comparatively favourable for an affection with turbid but sterile spinal fluid" will find general acceptance. Very little is said about the chemistry of the fluid in these conditions, beyond the statement that the chlorides are greatly reduced in tuberculous meningitis. Nonne's 'compression syndrome' and its confirmation by the 'Queckenstedt symptom' receive some attention, and the remainder of the chapter deals with the diagnosis of neurosyphilis.

In the appendix on encephalography, the insufflation of air by the lumbar route is fully dealt with, but Dandy's ventriculography is barely mentioned; in that on cistern puncture the names of Ayer and his co-workers do not appear, nor is any mention made of the injection of lipiodol; and in the bibliography, eight pages long, the only recent Anglo-saxon paper referred to is that of Dandy. Such wholesale exclusion of all but Teutonic literature is excusable in an Austrian publication, but might have been remedied in an English translation. The translator has followed the German idiom too closely for clearness, and in some places it is quite impossible to understand the author's meaning. Several decimal points are misplaced, mis-translations such as 'inspissator' for a thermostat at blood heat (Wassermann bath) are not uncommon, and words unfamiliar to English readers, such as 're-infundibulated', often occur. The book would have been improved by freer translation and more careful editing.

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**The Advance of Orthopædic Surgery.** By A. H. TUBBY, C.B., C.M.G., M.S., F.R.C.S., F.S.A., Consulting Surgeon to Westminster, Royal National Orthopædic, Evelina, and Christ's Hospitals, etc. Reprinted from the *Clinical Journal*. Crown 8vo. Pp. xii + 144, with 31 illustrations. 1924. London: H. K. Lewis & Co. Ltd. 7s. 6d. net.

THIS little book forms light and pleasant reading, and is at the same time a useful compendium of recent work, selected, perhaps, rather at random, and not really strictly confined to the limited period since 1912 that the author mentions in his first chapter. The short annotations on congenital deformities of the spine and spondylolisthesis and those on growth deformities (Legg's, Kohler's, Osgood-Schlatter, and König's diseases) are of special interest. In the chapter on static and postural deformities, Mr. Tubby's views on the importance of asymmetry in scoliosis are described at length; it cannot, however, be said that these are generally accepted by orthopædic surgeons, and this chapter should be read in a critical spirit. The physiological problem of the nature and innervation of plastic tonus and its importance in postural deformities is omitted; this applies also to the physiology of spastic conditions and the question of sympathetic innervation of muscles. A critical review of recent advances in orthopædics might be expected to contain some reference to these and other physiological problems. The physiological principles involved in tendon transplantation are also worthy of consideration, but are ignored; for example, Mr. Tubby would advise the transplantation of a part of the triceps into the biceps; that is, of a muscle into its direct antagonist, a method considered unsound by those who like to transplant muscles into their synergic assistants.

At the end of the book the principles of re-education are briefly mentioned, and a few paragraphs devoted to the question of the organization of orthopædic work and of the care of cripples. This important side of orthopædics is now receiving much attention from public authorities, and great progress is being made owing to the efforts of private individuals and others.

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**Tumours of the Spinal Cord.** By CHAS. A. ELSBERG, M.D., Professor of Neurological Surgery, Columbia University. Royal 8vo. Pp. 421 + viii, with 354 illustrations. 1925. London: H. K. Lewis & Co. Ltd. £2 10s. net.

ELSBERG's book is based upon a study of 81 cases of spinal-cord tumour upon which he has operated. An interesting historical introductory chapter is followed by some 230 pages of detailed case reports, which constitute about 60 per cent of the entire volume. The symptomatology, morbid anatomy, and treatment are clearly discussed in the succeeding chapters. The operation of laminectomy which he performs is, in all essential respects, that which was employed by Victor Horsley, and he rightly deprecates the cramping osteoplastic operations and 'hemi-laminectomies' which have from time to time been advocated by other surgeons.

The fairly common and very definite condition of meningitis circumscripta serosa which Horsley described is only briefly alluded to, and it is somewhat surprising that in so large an experience Elsberg should attach so little importance to this condition as a cause of compression paraplegia. Sicard's lipiodol-radiographic method of localization scarcely receives the notice which it deserves.

The book is an excellent work of reference, is well illustrated, and contains a good bibliography.

**Surgical Treatment of Pulmonary and Pleural Tuberculosis.** By J. GRAVESEN, Copenhagen. Pp. 155, with 87 illustrations (3 in colour). 1925. London: John Bale, Sons & Danielsson. 10s. 6d.

To those who are interested in the surgical treatment of advanced phthisis this book will prove useful, as it goes carefully into the question of the indications and contra-indications for the developing of artificial pneumothorax, the choice of the methods in collapse therapy, the details and advantages of thoracoscopy, and the operation of extrapleural thoracoplasty.

The book is well illustrated and well printed, and should serve a useful purpose.

**Die Chirurgie der Brustorgane.** By FERDINAND SAUERBRUCH. Vol. II. Pp. 1075 + xxxi, with 720 illustrations. 1925. Berlin: Julius Springer. G.M. 258 (= £12 18s.)

The first volume of this work was published in 1921. It contained the anatomy and physiology of respiration, the general pathology of diseases of the thorax, diagnosis of thoracic diseases, methods of anaesthesia, the uses of pressure chambers, and the general technique of thoracic surgery. Now Sauerbruch brings his work to completion by the publication of his second volume, a large book of 1075 pages and 720 illustrations, many of them in colour. This volume deals with the surgery of the heart and pericardium, the large blood-vessels, the mediastinum, the oesophagus, diaphragm, and breast, and included in it is the second edition of the *Technik der Thoraxchirurgie* originally published by Sauerbruch and Schumacher in 1911.

The book is a landmark in the literature of thoracic surgery, and the greatest credit must be given to the author for his painstaking work in pioneer surgery, and for the energy needed to produce such a work unqualified praise is due.

**Traité pratique d'Orthopédie.** By Dr. G. POTEL, Professor at the Faculty of Medicine at Lille. Royal 8vo. Pp. 734, with 399 figures in the text and 4 coloured plates. 1924. Paris: Gaston Doin. Fr. 80.

We have thoroughly enjoyed the reading of Professor Potel's work, although the enjoyment has been at times tempered by disappointment at the small space he devotes to treatment. Surgical proceedings of every-day occurrence are dismissed with a curt account, which would afford a very poor guide to any surgeon who wished to perform them. For example, only eleven lines are devoted to the description of Albee's operation on the spine.

Professor Potel wishes to exclude from his work all obsolete methods and all methods still in the experimental stage, and in this he is right; but he also dismisses in a few words methods which can claim a very fair amount of success, such as Stöffel's operation for spastic paralysis.

To pass from adverse comment to real appreciation. With typical French thoroughness and painstaking, Professor Potel has given us, if not the first, at any rate the best scientific classification and pathology of congenital deformities. An immense amount of patient observation and collection must have been needed for this. His explanation of certain congenital deformities such as 'torticollis' and 'club-foot' by a theory of relative muscular insufficiency or dystrophy appeals to us, and seems to explain the observed phenomena better than other theories. Especially does it explain the tendency to relapse after efficient correction as growth proceeds, and emphasizes the necessity for constant supervision for many years. Amniotic bands are rightly relegated to a place of minor importance as causative factors. The varieties of spina bifida and meningocele are well and accurately described, and are illustrated by excellent coloured diagrams. As we have mentioned above, spastic paralysis is ably but too briefly dealt with.

The second part deals with acquired deformities. Postural deformities of the spine are well described. Professor Potel takes a modern and, as we think, correct broad view of their causation, without giving to habitual faulty attitude an importance greater than its deserts. Pott's disease has been well studied in France, and is, as we should expect, well described, including the treatment, with the exception of treatment by operation.

For the rest, ununited fractures, ankylosis of joints, etc., are treated at length,

and we are pleased to see these subjects included in a book on orthopædies. Professor Potel's views we think excellent, especially his evident dislike of the use of foreign, metallic bodies for bone fixation.

On the whole the book is a very pleasing one, written in excellent style, and occasionally witty; we think that Professor Potel, having done so much, would be well advised to expand the sections devoted to treatment in the next edition, and give us a work of standard value from cover to cover.

*Patologia e Chirurgia della Milza.* By Professor L. SILVESTRI. Large crown 8vo. Pp. 402, illustrated. 1924. Bologna: Capelli. Lire 35.

THIS most interesting manual covers the ground of splenic pathology as required by the surgeon. It opens with chapters on the anatomy, physiology, and pathology of the spleen; passes to a description of misplacements, ruptures, and wounds; discusses the enlargements associated with syphilis, tuberculosis, malaria, and leishmaniasis; and takes up the study of cysts, vascular tumours, and malignant neoplasms, giving adequate descriptions of the conditions based on wide reading.

When it comes to dealing with the difficult and important subject of the splenic anæmias, the author, for all his industry, cannot get beyond the region of nice distinctions without essential differences. He has sections on the infantile form of Cardarelli-Jaksch, on the type Griesinger-Banti, on Banti's disease proper, on chronic icteric splenomegaly, on splenomegaly hæmolytica; but is forced to admit the encroachment of the one type on the other, and whilst recognizing the clinical value of splenectomy in all, rationalizes the procedure on seemingly contradictory hypotheses. In this he is, of course, not to blame, for the functions of the spleen are imperfectly understood, and vary at different age periods, in different phases of metabolic instability, and under the influence of noxa at present merely guessed at. From the study of this group he passes to the allied condition of pernicious anæmia, purpura hæmorrhagica, and so on. There is an absence of discussion of cases in which the pancreas must be included in the syndrome.

A third part is devoted to surgical procedures, describing, besides splenectomy, the various partial substitutes for removal.

The style is easy, and clearly the work is that of a man who is not only deeply interested in the clinical aspect of the subject, but also has that intimate acquaintance with the underlying pathology which comes of devotion to its experimental investigation. There is an extensive bibliography.

*Die örtliche Betäubung.* Von PROFESSOR DR. HEINRICH BRAUN, Gen. med. Direktor des Krankenhauses in Zwickau. Seventh edition. Medium 8vo. Pp. 511, illustrated. 1925. Leipzig: J. A. Barth. R.-m. 22.50.

THIS new edition of Braun's well-known work has been brought thoroughly up to date. It is, perhaps, the most reliable and authoritative book on the subject. The section dealing with local anæsthetic methods in operations upon the neck has been completely re-written. Very interesting is the chapter on abdominal surgery. Here the author's remarks are well worth attention. He maintains that general anæsthesia plays only a small part in causing post-operative pulmonary complications, the proportion of which is not greatly lowered by the use of local anæsthesia. Yet there are special indications, and on occasion local anæsthesia is the method of choice. The author does not advocate the paravertebral procedure, partly because so much novocain must be injected, partly because it is difficult always to ensure that every one of the eighteen to twenty-two nerves has been reached by the anæsthetic, partly because it imposes rather a severe strain upon both patient and surgeon. Splanchnic anæsthesia Braun regards as a great advance in abdominal methods. He employs the anterior route after opening the abdomen, but admits that in some cases a short general ethyl-chloride anæsthesia is necessary to carry out the splanchnic procedure. English surgeons as a body will probably not be inclined to agree with him that the problem of local anæsthesia for operations in the upper abdomen is solved by splanchnic methods. The rest of the book fully maintains the high standard reached in previous editions. To those interested in local anæsthesia there could be no better guide.

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## EPONYMS.

By SIR D'ARCY POWER, K.B.E., LONDON.

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### XVIII. BRYANT'S ILIO-FEMORAL TRIANGLE.

THE name of Mr. Thomas Bryant is associated with a pair of torsion forceps, a splint, and a surface-marking. The torsion forceps has gone into the limbo which sooner or later receives all surgical instruments. The splint is less used than it was. The diagnostic value of the ilio-femoral triangle remains. The first description of it appears in the course of a clinical lecture delivered at Guy's Hospital on December 16, 1875 (*Lancet*, 1876, i, 119), though attention seems to have been drawn to it at a meeting of the Royal Medico-Chirurgical Society on February 9, 1875.

Mr. Bryant introduced his subject with the following words: "The interest that is attached to the subject of injuries of the hip-joint, the difficulty that occasionally attends their diagnosis, and the injury that is too often inflicted upon a patient in the attempt to make out a difficult case, are some of the reasons that have induced me to bring before your notice on the present occasion a means of diagnosis in these cases that I have long employed and taught, although I may not have formulated it before the present year.

"I have described the means in this paper as the ilio-femoral triangle, and I have done so because the triangle is formed between the ilium and the femur. The lines which form it are readily made out and any shortening of the one which I have called the base, and to which I am about to draw your especial attention, can be easily detected.

"The triangle is formed as follows, and is a right-angled triangle. It is figured below (*Fig. 120*). One side of the triangle is represented by a line A B, drawn from the anterior superior spinous process of the ilium to the top of the trochanter major. The second, A C, is drawn from the anterior superior spinous process of the ilium directly downwards to the horizontal plane of the recumbent body. And the third, C B—which is the base of the triangle—is drawn at right angles to A C and falls upon the line A B, where it touches the great trochanter. It is to this line my observations refer. The line A B it will be seen corresponds in part to Nélaton's well-known line, which is



drawn from the anterior superior spinous process of the ilium to the most prominent part of the tuberosity of the ischium. This line in the normal position of the head of the femur, touches the upper border of the trochanter major in every position of the limb, and I believe that if this line is to be considered to be the test-line for dislocation of the head of the femur backwards—which I take it to be—I must claim the base of the triangle I have described (CB) to be the test-line for fracture or shortening of the neck.

“At any rate I can confidently assert, after repeated proofs, that whilst in a healthy subject these ilio-femoral triangles are exactly similar upon the two sides of the body, with equal sides and equal angles, I can with equal confidence assure you that in all cases of injury to the hip in which shortening of the neck of the thigh-bone exists, the amount of shortening can readily and accurately be made out on comparing the bases (CB) of the triangles of the two sides. That in impacted fracture of the neck of the thigh-bone, where on the sound side the base of the triangle will, in the adult, measure its average normal length of two and a half inches, on the affected or injured side it will measure from half an inch to more than one inch less. These

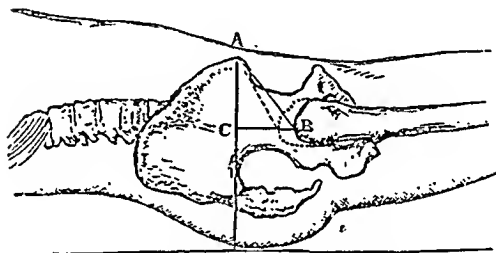


FIG. 120.—Bryant's triangle.  
(Figs. 120 and 121 are facsimiles of the original illustrations in the *Lancet*.)

measurements being taken with the patient in the horizontal position, the pelvis straight, and the two femora parallel.”

He then gives details of six cases of injury to the hip in which the ilio-femoral triangle had been useful in making a diagnosis, and adds that “in practice the line is easily made out and the test as to shortening of the neck of the femur readily applied, and with these facts before us the value of the means for diagnostic purposes can hardly be disputed. By means of this line the diagnosis of an impacted fracture of the neck of the thigh-bone can, as a rule, be made out with facility and certainty; and in a large number of obscure cases of injury to the hip the doubts and difficulties that were formerly experienced may be exchanged for the confidence of accurate knowledge. I do not mean, however, to say that by means of this test-line all obscure cases of injury to the hip-joint can be cleared up, for such an assertion would not be true; but I would wish you to believe what I have found to be the case—that by its use a large number of cases that would have been called obscure have ceased to be so. It is true there may be some shortening of this line in cases of fracture of the great trochanter, and it is likewise possible that there may be *no* shortening of the line in some examples of impacted fracture; but these cases are exceptional, and they in no way tend to diminish the diagnostic



THOMAS STUART, D.D.  
"Staple for Boston"  
1877-1878



value of the means I have to-day brought before your notice in the majority of cases of fracture of the neck of the thigh-bone, and in all cases in which shortening of the neck of the femur may be found. With these remarks I respectfully commend this test-line to your notice and adoption."

Mr. Bryant continues: "I think I shall be able to show that the opinion I have already expressed concerning the value of the test-line has been satisfactorily proved to be true by the experience of my dressers in the wards of this hospital; for I can tell you, and with much satisfaction, that during the last three years at least sixteen consecutive cases of fracture of the neck of the thigh-bone have been admitted into my wards, and that all these sixteen left with good union of the broken bone and useful limbs."

### TORSION FORCEPS.

"When in his prime", says a writer in the *British Medical Journal*, "there was much discussion and experiment in relation to the question of hæmostasis without ligatures. Charrière in 1858 introduced his catch forceps with a lock and ring handles like scissors, devised both for dressings and for hæmostasis by torsion. Two or three years earlier Weber had invented an anti-ligature forceps, which, however, never came into wide employment. Koeberle, a pupil of Spencer Wells, made use of Charrière's forceps for forcipressure, maintained for several hours or days, in 1862, and continued the practice until 1867, when he removed the pressure forceps at the end of the operation or sooner, as is now the almost universal practice. Péan in 1867, and Wells about 1872, brought forcipressure into general use. Bryant's *Practice of Surgery* appeared in that year, and the author strongly advocated in it the method of torsion, figuring the instrument still known as Bryant's forceps. The instrument was thus made known two years before the publication of that which is usually described in maker's list as 'Wells' forceps.' In 1874, however, Wells' forceps were used as much or more for torsion as for forcipressure, though on the other hand we must remember that it was first made for Spencer Wells in 1872, the year that Bryant's text-book first appeared". Mr. Bryant stated in the fourth edition of his text-book that in 200 consecutive cases of amputation of the thigh, leg, arm, and forearm, in all of which the arteries had been twisted and not tied, there had been no case of secondary hæmorrhage, and this at a time when nearly every wound supplicated. In no fewer than 110 cases it was the femoral artery which had been twisted.

### BRYANT'S SPLINT.

Bryant's splint (*Fig. 121*), described in the *Lancet*, 1880, i, 159, was intended to keep the legs and thighs quite parallel and to obviate the necessity for a perineal band. It was a modification of De Morgan's splint, and consisted of two long wooden splints interrupted from a point below the great trochanter to just above the crest of the ilium, the interruption being designed to avoid pressure upon the hip. One splint was applied to the outer side of each limb, and the two were connected below the feet and over the chest by sliding rods which could be fixed at an appropriate width. Extension was maintained

by an accumulator band, and counter-extension was obtained by the weight of the patient's body when the foot of the bed was raised three or four inches by blocks placed beneath the feet. The splint served a useful purpose, for it was introduced at a time when much thought was being given to the treatment of tuberculous disease of the hip by physiological rest.

Mr. Bryant's life history was told sympathetically by Mr. Golding Bird in the sixty-eighth volume of the *Guy's Hospital Reports*. He was born in 1828, the son of Thomas Egerton Bryant, who practised at Kennington and was surgeon to the Lambeth Infirmary. His father had been educated at the United Borough Hospitals, was interested in surgical pathology—as became a pupil of Sir Astley Cooper—and was President of the Medical Society of London in 1837. He sent his son to King's College School, apprenticed him to Mr. Thomas Olliver Duke, of Herleyford Place, his successor as Surgeon to the Lambeth Infirmary, and entered him in 1846 at his old school at Guy's Hospital five years after the death of Sir Astley Cooper. Thomas Bryant obtained the M.R.C.S. Eng. in 1849, and was elected a Fellow of the Royal

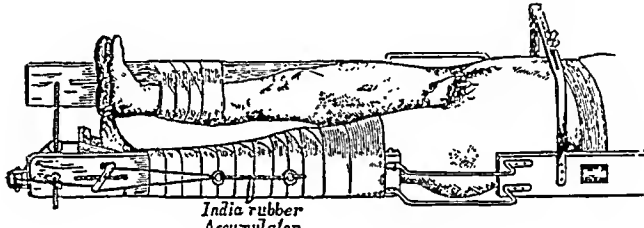


FIG. 121.—Bryant's splint.

College of Surgeons of England after examination in 1853. For some reason he never served as a demonstrator of anatomy as was then usual for those who aspired to the surgical staff of the hospital, but he was elected assistant surgeon in 1857, though he did not become full surgeon until 1871. He retired from the active staff in 1888 on reaching the age of 60, and having few distractions outside his profession he became surgeon to the Bolingbroke Hospital.

In addition to his hospital appointment he had a long and honourable connection with the Royal College of Surgeons, acting as Examiner, Member of Council, and President. In 1893 he was chosen to deliver the Hunterian Oration on the occasion of the hundredth anniversary of the death of John Hunter, the oration being given in the presence of H.R.H. The Prince of Wales, afterwards H.M. King Edward VII, and his son the Duke of York, afterwards H.M. King George V. In 1896 he was appointed Surgeon-Extraordinary to Queen Victoria, and later Surgeon in Ordinary to H.M. King Edward VII.

He published in 1872 a *Manual for the Practice of Surgery* which had a large sale in the United States as well as in this country. The book was essentially clinical, and was written in an easy style which made it especially acceptable to those who did not intend to practise surgery as a specialty. He also wrote an excellent clinical book on the *Diseases of the Breast*, consisting

of material drawn chiefly from his own experience. It appeared in England in 1887, and was published separately in New York in 1889.

He died on December 31, 1914, having married in 1862 Adelaide Louisa, daughter of Mr. Benjamin Waldron, whom he survived three years and by whom he had four sons and three daughters.

Mr. Bryant, like his contemporary Sir William Savory, was unable to appreciate the great work which was being done by Joseph Lister. Bryant and Savory were both clinical surgeons without real scientific training. Both were wedded to the methods which they had practised for many years and by which they obtained better average results than some of their colleagues, partly owing to their greater operative skill, and partly by the attention which they bestowed on the after-treatment of the patients upon whom they had operated. Both, therefore, had attained the dangerous mental attitude of believing that surgery had attained to perfection.

Cheery, optimistic, and of pleasant manners, Bryant soon attained a very large surgical practice, driving about the town in a private hansom cab when he was very busy, in a one-horse brougham when there was less to do, and in a carriage and pair when business was slack. There were fewer operations for London surgeons, and no nursing homes for their patients, in the middle of the last century, and the professional work of a successful consulting surgeon lay within the compass of a horse drive, except for an occasional railway journey.

The portrait is copied from the oil painting by M. Hickson which hangs on the grand staircase at Guy's Hospital, and is a good likeness.

marked posterior kyphotic curve in the cervical and upper dorsal regions, and an anterior curve of the lower dorsal and lumbar vertebræ. Its length decreased, and his height sank from 6 ft. 1 in. to 5 ft. 9 in. The chest, owing to lateral flattening, became narrow, while its antero-posterior depth was increased. The posture was striking and simian. The head was advanced and lowered so that the neck was very short, and the chin more than 1 in. lower than the top of the sternum.

The short narrow chest suddenly widened into a much shorter and broad abdomen, and the pelvis was wide and low. The arms appeared unnaturally long, the hands hanging low down by the thighs and in front of them.

During the last three or four years of life the increase in the limbs and head was imperceptible. There was no disturbance of the general health, and the upper limbs remained perfect until in January, 1876, a swelling of the upper third of the left radius appeared and grew rapidly. The patient gradually failed and emaciated, and died on March 24 with pleural effusion due to metastatic growths in the right pleura, and nodules of growth in the vault of the skull. Except for the primary growth in the left radius the rest of the bone seemed quite healthy.

**Dr. Wilks' Case.**<sup>3</sup>—A man, aged 60 at the time of his death, had sought advice fourteen years before for pains in his legs. His tibiae soon became enlarged, and after two years it was evident that all the bones in his body were increasing in size. The humerus, clavicle, and cranium were all involved, and subsequently the ribs.

A year before his death his health began to suffer from the implication of the thorax, and from this time it was apparent that his days were numbered. "At this period he was accustomed to sit in his chair with his head bent forward and chin resting on his breast, his arms and legs almost useless, and his breathing very difficult from want of expansion of the chest."

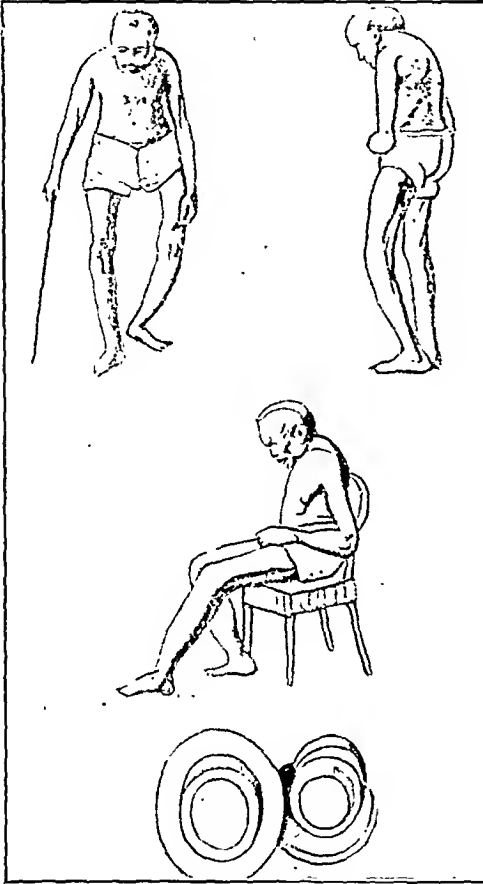


FIG. 122.—Reproduced from Paget's original paper (Gaenslen).

resembled much more that of an ape than of a man. He had become shorter in stature, his legs were somewhat bent, his arms fell in front of him, his head dropped forwards, his chest was narrowed, pelvis contracted" (described by Dr. Goodhart—p.m.—as of the rickety type) "and belly protuberant, and thus all characteristics of a manly figure lost."

"After this the chest became more contracted, and at last quite fixed, so that the respiratory process was effected entirely by the diaphragm. Breathing became painfully difficult and dropsy of the legs ensued . . . the breathing became more difficult until at last the respiratory apparatus altogether stopped."

A cauliflower growth (endothelioma of the arachnoid) was growing from the inner surface of the dura mater, and pressing into but not involving the brain.

In some cases the incidence of the disease does not follow the usual course. Clutton<sup>4</sup> recorded the case of a woman, in whom, though the usual bones were affected, the disease reached its height in the upper extremities; and Joseph Griffiths<sup>5</sup> has described a skeleton in the Cambridge Museum which had the same peculiarities.

Wherry<sup>6</sup> amputated a leg for sarcoma of the tibia in a man, age 56, in whom the skull and vertebral column were the parts chiefly affected.

The long bones were not obviously diseased in Goodhart's case<sup>7</sup> of the old post-boy, but the skull was remarkably affected, and the clavicle, 1st rib, spine, and pelvis were involved.

Possibly Paget's doubt as to Rullier's case<sup>8</sup> was due to the fact that the bones of the limbs were normal and appeared small and disproportioned when compared with the other parts.

Lastly, one of J. R. Lunn's cases<sup>9</sup> (*Case 3*) had ankylosis of both hips and stood with crossed legs. This was almost certainly due to complications and not to osteitis deformans. The joint troubles were unusually marked.

The type in which *one bone only* is affected is more frequent than published cases would suggest. Many surgeons believe that in these cases trauma is more often the exciting cause than in the generalized type. A history of a contusion or a fracture antecedent to the onset of the disease is common, but it is not always to be obtained, and as the interval between the injury and the commencing deformity may be considerable the point is a difficult one to settle. The best known instance of this limitation of the disease is that recorded by Bowlby<sup>10</sup> :—

A cabman, age 64, died in St. Bartholomew's Hospital from fractured skull and cerebral hæmorrhage. His left femur presented the usual signs of the disease. It was curved in an outward and forward direction, and very flattened antero-posteriorly. Its circumference at its thickest part was  $6\frac{1}{2}$  in. Its surface was rough and uneven, and the neck was at right angles to the shaft. It was the only bone affected, and had become curved during the last ten years of life. A few months before death the shortening amounted to  $2\frac{1}{2}$  in. (*Figs. 131, 131A*)

The following case was associated with trauma and complicated with ulcer and necrosis, conditions which will be subsequently considered :—

J. R., age 63, broke his right leg thirty-four years before admission to the Leeds General Infirmary. For a good many years following the fracture walking had caused great pain, and for the last twenty years the leg had been slowly bending. He had had to give up his work nine years ago, and after that the leg became swollen, and an ulcer formed which never healed. The limb was amputated by Mr. Littlewood. The tibia was the only bone affected.

It is the bone changes that constitute the important and characteristic feature of osteitis deformans. One symptom—pain—is directly connected with them.

*Pain* in the bones is usually marked long before any alteration can be detected in them. It continues during the progress of the bone changes and varies in intensity. In some cases it is not present at all, or is insufficient to attract attention. Joncheray has distinguished a painful and a painless



variety, and believes that the osseous lesions develop more rapidly in the former. Hurwitz has described two cases of the painless form (*Cases 2 and 4*) in which the disease developed marked features unsuspected by the patients, who came under observation for the results of cardiac and vascular trouble and not for any condition arising from the bone disease.

*The period of life* in which the disease begins is a point of considerable importance. It is rare for osteitis deformans to start before the age of 40, but earlier cases have been recorded. In Morris's case the symptoms began at 30, and in Paget's *Case 7*, a woman, as early as 28.<sup>11</sup> On the other hand, Packard states that out of 41 males and 24 females, the youngest reported was Watson's at 39. One or two cases have been met with below the age of 20, but such cases should be looked upon with suspicion, and only accepted with caution. They fall within the period of life when osteitis fibrosa develops, and there has been much confusion between the two diseases. I recently examined a section from a clavicle removed from a girl of 19 which had long been regarded as an instance of osteitis deformans. The microscopical appearances were those of osteitis fibrosa (Leeds Medical School Museum, old No. A119b (*see p. 231*), new No. A814).

On the other hand, Mr. P. B. Roth recently reported to the Orthopædic Section of the Royal Society of Medicine the case of a man of 30 whose right tibia had been growing bigger since the age of 18. The skiagram, shown at the meeting, was typical of osteitis deformans. The tibia was the only bone affected.

## 2. THE BONE CHANGES.

Enlargement and softening characterize the bones which are attacked by osteitis deformans. The long bones are not only greatly thickened, their circumference and sectional area being much enlarged, but they are also elongated and unduly curved. The alteration in the normal curves, not only of the bones of the lower extremities but of the spine, is due to the superincumbent weight acting upon bone softened and rarefied by the disease. In the upper extremities, sometimes, different but not very dissimilar causes are at work. In some instances bending is produced by the elongation of a bone attached at both ends to another bone which has not been affected. This happens in the forearm and the leg. Notwithstanding the elongation of the long bones, a decrease in height is a constant result of osteitis deformans. It may vary from a few to  $13\frac{3}{4}$  inches (Hurwitz.<sup>12</sup> *Case 5*), and depends chiefly upon the curving of the spine and of the bones of the lower extremities.

## THE SKULL.

It is exceptional for the skull not to be greatly enlarged. The increase in size is at first associated with great vascularity, but this slowly wanes as sclerosis develops. Three typical stages can be recognized.

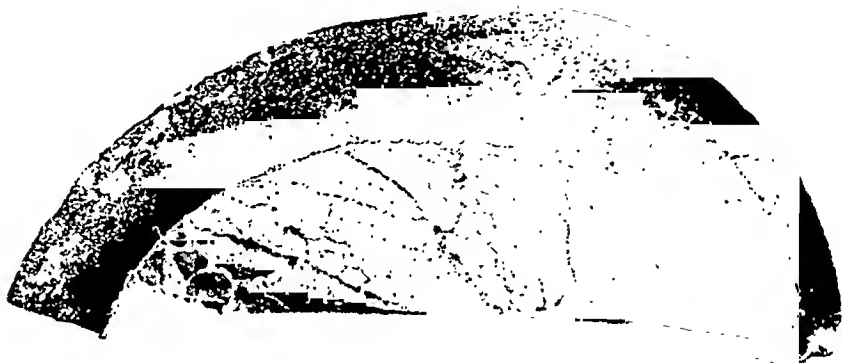
1. **The Vascular Stage.**—In this the bone is of a marked red colour due to the permeation of the new porous bone from side to side by a very vascular connective-tissue marrow. It was present in the cranium of a woman, age 65, who had suffered constant pain in the head—often severe—for fourteen years. Its circumference had increased nearly an inch (to  $25\frac{7}{8}$  in.) during the



FIG. 123.—The vault of the skull of Dr. Hann's case. Miss Wright has given a very good representation of the present colour of the skull. When fresh the red colour was more pronounced. (No. A 26n, Leeds Medical School Museum.)

four years she was under observation. The vault is still of a brick-red colour, testifying to its great vascularity; and owing to its softness it was removed with great ease. Condensation on the inner and outer surfaces is in an early stage, but not differentiated in colour from the open porous tissue which constitutes the bulk of the calvarium. The sutures are not obliterated and the frontal sinuses persist. Microscopically the vascularity was very evident. (*Fig. 123*). (Leeds Medical School Museum, No. A26n.)\*

**2. The Stage of Advancing Sclerosis.**—Many museum specimens illustrate this stage. The skull of Paget's case is a good example (R.C.S. Museum, No. 1239) (*Fig. 124*). In it the thickness of the vault in every part is four times the normal, and the sutures of the vertex are obliterated. Its outer surface is smooth, but perforated by multitudinous minute apertures. It is the surface of very finely cancellated porous bone. The inner surface is similarly



*FIG. 124.*—Sagittal section through the cranial vault of Paget's case. Note the sclerosed condition of the inner part of the section and the islet of condensed bone on the right. (No. 1239, R.C.S. Museum.)

but less obviously porous. It is deeply grooved for meningeal vessels, and perforated by numerous vascular foramina. The general character of the interior of the bones, whether of the vault or base, is a finely porous or reticulate condition differing in degree and uniformity in different parts. A section through the vault shows the bone contiguous to the inner surface to be very condensed for a thickness of  $\frac{1}{4}$  to  $\frac{1}{2}$  in., but it is still finely porous. (Paget described it as "hard white-looking bone, dense like limestone".) The rest of the section shows various degrees of porous, cavernous, or cancellous tissue whose spaces were filled with soft reddish substance, whilst the outer surface is bounded by a thin line of condensed porous bone of the thickness of paper.

**3. The Stage of Complete Diffuse Sclerosis.**—This is exemplified by the cranial vault of Dr. Goodhart's case (R.C.S. Museum, No. 705.1, Gen. Path.

\* The case, which is of much interest, was recorded by Mr. R. G. Hann (*Brit. Med. Jour.*, 1910, i, 135). The patient died with cerebral symptoms, but no gross lesion was found in the brain. The botryoidal elevations on the inner surface of the vault are not commonly of such a size.

Sec.) (Fig. 125). The sclerosis has advanced across the diploic zone and invaded in an irregular and patchy manner the finely porous tissue which may be considered to represent a thickened external table. In the vertex, but not at the sides, the cancellous diploic area is still recognizable where it is not crossed by sclerosed tracts, but it is very narrow. The specimen presents an ivory-like appearance with a white and somewhat polished exterior (*see also* No. 1241Bk, R.C.S. Museum—another example). On the internal surface a small opening leads into a cavity big enough to hold a damson, and occupying almost the whole thickness of the skull.

In skulls that illustrate the last two stages, *sharply defined islets of ivory-like bone* may frequently be seen on the face of the sections isolated in a



FIG. 125.—Showing complete sclerosis of the cranial vault. There is an islet of condensed bone near the top of the picture, which looks as if it were in process of separation. Goodhart's case. (No. 705.1, R.C.S. Museum, Gen. Path. Sect.)

cancellous area or surrounded by a thin line of porous tissue in a sclerosed part. Some are defined by what is suspiciously like a line of demarcation, which suggests that they may eventually become sequestra. Possibly the cavity in the vault of Goodhart's case may at one time have contained a sequestrum which became absorbed during the long duration of the disease.

THE FRONTAL SINUSES are usually found open when the section through the skull passes across them. They tend to become smaller or even obliterated in advanced cases, but this tendency is not so marked as in both varieties of leontiasis.

THE CRANIAL CAVITY is not clearly diminished in these hypertrophied skulls, the increase in the thickness of the bone taking place almost entirely outwards.<sup>13</sup> The earlier sclerosis of the inner portion of the vault may be

explained by the compression of its interstitial substance owing to the resistance of the cranial contents to the inward expansion of the enlarging bone.

THE BASE OF THE SKULL may appear quite normal in some cases, but in a fair proportion it is obviously involved. Then the bones appear swollen, the grooves for the meningeal vessels are deep and sharply margined, and the pituitary fossa may be altered. The various foramina are not seriously contracted, though the regularity of their margins may be impaired. This is most noticeable in the foramen magnum (R.C.S. Museum, No. 1241 Bk). Wyllie,<sup>14</sup> commenting on this narrowing of the neural foramina and the rarity with which the cranial nerves show signs of compression, records two cases in which optic atrophy very possibly depended upon compression of the nerves at the optic foramina. On the other hand, the vascular foramina have occasionally been found enlarged, presumably from the pressure exerted upon the softened margins by the pulsation of the transmitted vessels.

The anterior fossa in some instances presents a curious appearance. A deep V-shaped depression with shelving sides and its apex at the cribriform plate is produced by the great thickening spreading to the orbital plates from the frontal bone.

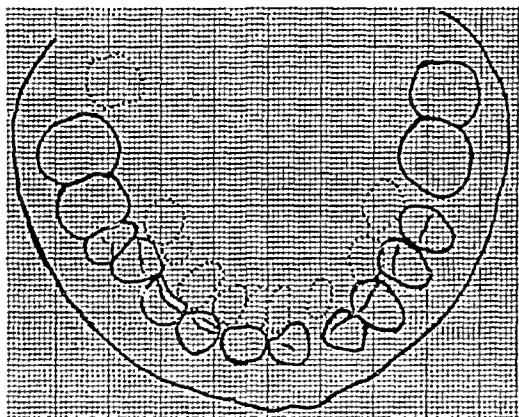


FIG. 126.—Diagram plotted from casts of upper and lower jaws from a case of osteitis deformans, showing the enlargement produced in the alveolar arch of the upper jaw by the disease, and the position of the alveolar arch of the lower jaw within it. (By the kindness of Sir Frank Colyer.)

the skull formed from membrane are more conspicuously affected than those formed from cartilage, but the relative proportions of compact and cancellous tissue are probably more important than the mode of origin. It is easy to realize that the petrous bone will resist absorption and expansion much more than bones with a thin cortex and a considerable cancellous interior.\*

THE FACIAL BONES are commonly thought to escape, and probably a

The softened cranial bones will give way to pressure like the long bones, and a suggestive asymmetry may result. In one skull, probably as a result of the position in which the head was rested, the posterior fossa on the right side was narrowed and elongated, and the internal occipital crest deviated to the left (St. Thomas's Hospital Museum, No. 422). In another the left posterior fossa was narrowed, whilst the parts about the foramen magnum appeared pushed up and produced a hollow on the under surface of the base (Univ. Coll. Hosp. Museum, No. 652, Bone 64H). It has been pointed out that those parts of

\* In the *Journal of Laryngology and Otology*, 1923, July, xxxviii, 344, G. J. Jenkins discusses deafness in connection with osteitis deformans. Nine cases with obvious affection of the skull bones suffered from deafness in some degree. He came to the conclusion that deafness is not obvious until the skull bones are affected.

slight involvement often evades notice. Sometimes, however, they are markedly affected (Univ. Coll. Hosp. Museum, No. 652, Bone 64H.)

In the case of an edentulous man who was so deaf that no history could be obtained from him, the alveolar processes of the superior maxillæ were greatly thickened and rounded, and the dental arch so much enlarged that the unaffected alveolar margin of the lower jaw fitted completely and easily within it, like the rim of a box inside its lid (*Fig. 126*).\*

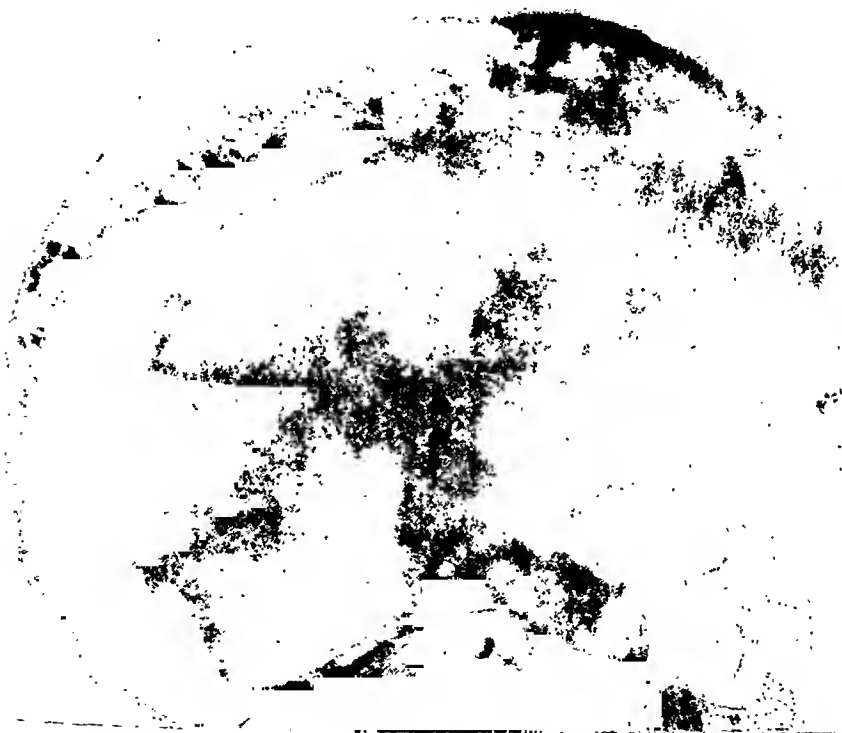


FIG. 127.—Skiagram of a skull of a man, age 54. The change in the skull was noticed twenty years before. (*From a case of Mr. C. P. G. Wakeley's.*)

\* Mr. J. G. Turner showed to me at the Royal Dental Hospital a retired policeman, age 62, who had first noticed the disease in the right tibia twenty years before. Both tibiae, the lower half of the right humerus, and the right clavicle were affected, and the size of the head had been increasing steadily (circumference 26 in.). The face bones were all enlarged—both maxillæ, the two maxillæ, and the mandible. The enlargement of the maxillæ was most marked, and when the edentulous jaws were closed the mandibular arch lay completely within that of the maxillæ. In addition to the general enlargement of these bones there was a circumscribed local enlargement of the molar region of the alveolus of the left maxilla. When the patient came under Mr. Turner's care both jaws were full of very foul teeth, and there was much pyorrhœa. The dental trouble must have existed many years, and probably dated back as far as the commencement of the bone disease. All the septic teeth, to which large masses of granulation tissue were adherent, were cleared out, and portions of bone removed from all round the upper alveolus, including the local enlargement, to lessen the deformity. Not only was the jaw condition improved by this, but so also were the tibiae, for the skin over these bones had been œdematous and inflamed and the bone tender, and this speedily cleared up.

In a man of 65 under the care of Sir Gordon Watson both upper jaws were considerably enlarged. The enlargement was general, and more marked on the left side. The body of the lower jaw was thickened. There was some dental trouble, but it was not severe. In both cases the jaw trouble was part of a generalized condition.

Cases in which the jaw enlargement would appear to have been only a local phenomenon are found in the literature of osteitis deformans. They are more likely to have been cases of osteitis fibrosa and probably dependent on dental irritation.

### THE SPINE.

The curving has been described in Sir James Paget's epitome.\* It is not infrequently associated with traces of 'lipping' of the bodies, with syn-

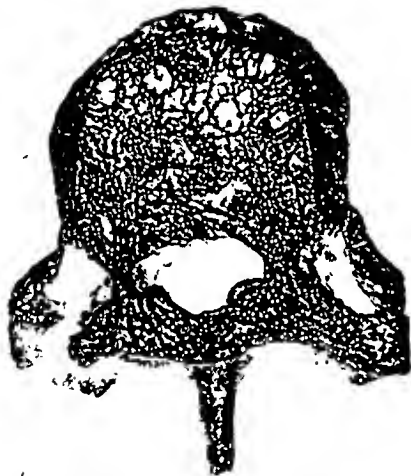


FIG. 128.—Section through a lumbar vertebra of a case of osteitis deformans showing: (a) Diminution and alteration in shape of the spinal canal by the enlarging body and neural arch; (b) The projection into its lumen of bony outgrowths from the arch; and (c) The formation of a lip around the articular edge, probably the result of pressure upon the softened bone. (College Stores, R.C.S. Museum.)

ostosis of their articular surfaces, and ankylosis of the dorsal spine—conditions apt to be readily dismissed as osteo-arthritis, but in which pressure, compensation, and senile changes in the discs undoubtedly play their part. Thickening, rarefaction, and softening of the various parts of the vertebræ take place, and occasionally one or more vertebræ may collapse and become deformed (R.C.S. Museum, No. 1241 Bf. 1241Bk: St. Bartholomew's Hospital Museum, No. 74Ci). Dr. Goodhart found a deposit of new soft bone all down the front of the bodies in the spine of the old post-boy, and in a skeleton in St. Thomas's Hospital Museum (No. 417) the bony deposit on the spines, articular and transverse processes, and also on the crests and dorsal surfaces of the ilia, approximates in its characters to myositis ossificans.

But a change that is little known, yet of great importance, is the diminished calibre of the spinal

canal which is brought about by the enlargement of the bodies and the bone composing the vertebral arches (Fig. 128). This may take place to such an extent that the cord and its membranes no longer lie loosely in the perithecal space, but come to fill the whole sectional area of the canal, and may even

\* I am indebted to Mr. C. P. G. Wakeley for an account of a man, age 51, in whom the history of the disease dated back twenty years. He sought advice for an abdominal swelling, which proved to be caused by the liver pushed forwards by a very pronounced anterior arching of the lumbar spine. (Fig. 127 shows the skull in this case.)

be subjected to compression with resulting paraplegia. In such cases additional localized pressure points may result from the formation of small osseous excrescences springing from the posterior surfaces of the bodies and other parts of the wall of the canal (Greenfield).

Two cases of osteitis deformans in which compression of the cord with paraplegia was brought about in this way have been put on record by Dr. W. G. Wyllie from the National Hospital for the Paralysed and Epileptic, Queen Square.

In one, a man of 55, with marked changes in the skull, spine, and one tibia, suffered from increasing weakness and sensory disturbances in both legs, with vesical and rectal inconvenience. Mr. Percy Sargent performed laminectomy of the 2nd to the 4th dorsal vertebral arches. The bone was thickened and soft, and cut easily with the forceps. The perithecal space was absent, and the inner surface of the laminae lay in close contact with the dura. Pulsation, which was at first feeble, improved as decompression was completed. The effect of the operation upon the symptoms was satisfactory. Sphincter power was regained, and the patient became able to walk much better.<sup>15</sup>

The other was a man of 63 who was admitted with complete spastic paraplegia which had been coming on gradually for two years. During an exploratory operation very similar conditions to those described in the preceding case were disclosed. At a subsequent post-mortem the spinal canal was found narrowed so that the dura mater was everywhere in contact with the thickened bone and also abnormally adherent to the arches all the way round. The bone disease appeared limited to the spine, but the condition of the long bones and the skull could not be investigated. It was in this case that Dr. Greenfield found that the cerebrospinal fluid, of which only a small quantity was obtained by lumbar puncture, was of a yellow colour and coagulated to a solid mass in the test-tube on standing (Froin's syndrome).<sup>16</sup>

### THE LONG BONES.

To appreciate fully a description of the macroscopic appearances of the long bones it should be realized that the morbid process consists in a complete or almost complete removal of the original osseous tissue and the substitution for it of a cancellous bone of a finely porous character. The piecemeal removal of the former, and the formation of the latter, are brought about by an active young connective tissue\* which permeates the trabecular framework of the bone from the fat medulla in the central canal (which often is only superficially involved) to the under surface of the periosteum, with which it blends.

This young connective tissue speedily shows that it has osteogenic properties. As the original bone disappears, new bone-formation begins in the osteogenic tissue which takes its place. The two processes—absorption and

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\* Though osteoclasts share in the removal of the original bone, as they certainly do in moulding the newly-formed bone, it is evident that the extensive destruction is out of all proportion to the number of osteoclasts present.



ossification—go on simultaneously, but the proliferating soft tissue forms first and slowly effects the expansion of the periosteal envelope during absorption, whilst the formation of new bone progresses more gradually. Thus soft and vascular tissue predominates in the diseased bones in the earlier periods of the disease.

Consequent upon these changes there ensues an excentric enlargement and



FIG. 129.—The upper half of a femur from Paget's case, showing its roughened and nodular surface. (No. 1240, *R.C.S. Museum*.)

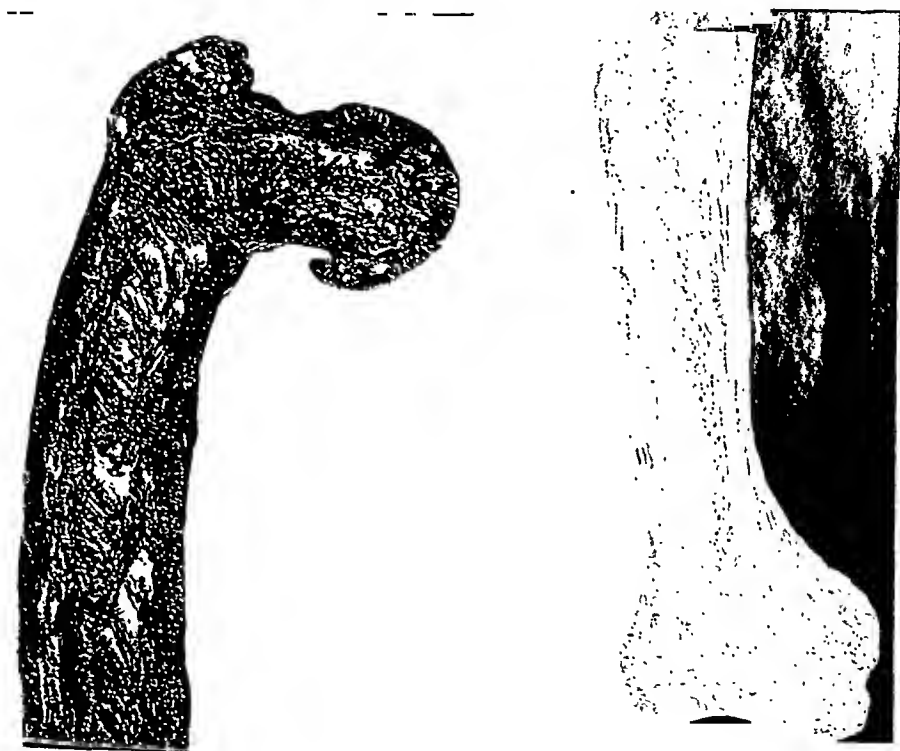
elongation of the bones, and a degree of softening, owing to the temporary inadequacy of the new bone-formation, which predisposes them to bend slowly under the influence of such forces as may be continuously or often in action. It is the bone, as distinct from the periosteum, that is involved in this process, and though the periosteum plays a part, it is not the subject of a true periostitis as that is generally understood. The periosteum limits the active tissue, shares in its nutrition, and no doubt assists in its superficial



FIG. 130.—This specimen may represent the stage of the disease which has been described in the text as the 'vascular stage'; but it is possible that the peculiar appearance may be due in part to atrophy from old age and disuse, and that the soft parts, instead of vascular connective tissue, may have been fatty. From a woman, age 73, who had been bedridden fifteen years and in whom the curvature of the tibia had been in progress thirty years. The head and right femur were also affected. (*Dr. J. R. Lunn's case*. No. 1241Bn, *R.C.S. Museum*.)

development: not by the laying down of superficial laminae, but rather by stimulating bone-production in the adjacent osteogenic tissue. The time arrives, however, when the ossification becomes more vigorous than absorption; the soft osteogenic tissue becomes largely turned into bone; and in the last stage the bones become dense, hard, heavy, and apparently strong.

The bone changes affect both the external and internal appearances. Paget, describing the long bones and their *normal* periosteum in his case, wrote: "The outer surface of the walls of the bones was irregularly and finely nodular as with external deposits or outgrowths of bone, deeply grooved with

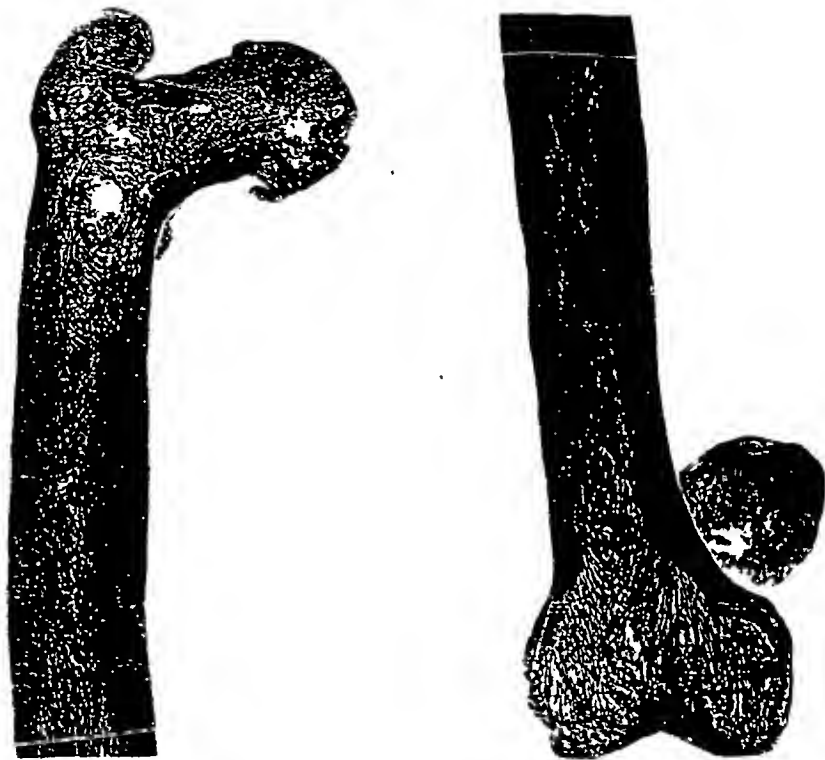


FIGS. 131, 131A.—Section through the femur of Bowlby's case—stage of advancing sclerosis. (No. 1241Ba, R.C.S. Museum.)

channels for the larger periosteal blood-vessels, finely but visibly perforated in every part for the transmission of the enlarged small vessels. Everything seemed to indicate a greatly increased quantity of blood in the vessels of the bone" (No. 1240, R.C.S. Museum, Paget's case) (Fig. 129). This irregular roughened surface of the long bones is very characteristic of the disease, and it will be noticed that Paget recognized that the irregularities might arise *from* the bone and not be laid down upon it.

The condition of the interior varies considerably, and, as in the skull, different stages may be distinguished. Thus in *some specimens* (macerated)

the interior of the bone shows a very open arrangement of trabeculae with irregular spaces between them, which were no doubt occupied by vascular connective-tissue marrow of a deep-red colour. The original compact bone has disappeared and a sclerosed outer layer of porous bone of no great thickness is present (Tibia, No. 1241Bn, R.C.S. Museum) (*Fig. 130*). In others the shaft of the bone is composed of thick walls of condensed bone enclosing a central cavity, much elongated and often irregularly enlarged. Its boundaries may be very unevenly sclerosed, and exhibit patches of rarefaction and many open spaces having a longitudinal direction as if separating vertical lamellae. In



*Figs. 132, 132A.*—Section through the femur of Paget's case. Stage of complete sclerosis. Owing to the marked anterior curvature of the bone, the section has passed close to the periphery of the central cavity in the middle portion. (No. 1240, R.C.S. Museum.)

many places these spaces are found close to the surface (Femur, No. 1241Ba, R.C.S. Museum, Bowlby's case) (*Figs. 131, 131A*). In a third series there is a uniform thickening and a dense sclerosis of the shaft enclosing a contracted elongated central cavity (Femur, No. 1240, R.C.S. Museum, Paget's case) (*Figs. 132, 132A*). The central medullary cavity is hardly ever so much altered as to be unrecognizable. In the femur of Paget's case (late stage) it is long, well-defined, and narrower than normal. In others it may show irregularities owing to absorption of adjacent cancellous areas, or it may be broken

up into irregular large compartments by transverse septa of some thickness (No. 1241Ba, R.C.S. Museum). In the tibia already referred to (No. 1241Bn), the central cavity is filled with a network of fine trabeculae simulating in delicacy the chordae tendineae of the heart, and mostly having a horizontal disposition. Quite definite changes are present in the *femoral head and neck*. The angle of the neck with the shaft is diminished in most cases—usually almost to a right angle. The cancellous tissue may be sclerosed (No. 1240).

The compact layer of the articular surface is apt to be of paper-like thinness, and may in parts be absent, a porous surface very slightly raised above the surrounding surface taking its place (Nos. 1240, 1241Ba, R.C.S. Museum). The stage reached in the neck does not necessarily correspond to that present in the shaft.

### CYST FORMATION.

The formation of cysts by liquefaction of the connective-tissue new formation as in osteitis fibrosa is very doubtful in osteitis deformans. The morbid process is characterized by organizing and not retrogressive changes. Bloodgood<sup>17</sup> suggests that such instances as have been recorded may all have been really cases of Recklinghausen's disease (osteitis fibrosa). I have not met with a single instance in the many bones I have examined in which the cystic nature of an intra-osseous space was beyond doubt. The cavity in the cranial vault of Dr. Goodhart's patient was the nearest approach to it. The spaces frequently seen in macerated bones have probably been occupied with osteogenic tissue, or in a few instances by granulation tissue or serum associated with the absorption of a sequestrum.

From the varied picture presented by the interior of the bones in Paget's disease, it is clear that absorption of the original bone and the formation of new is the dominant feature. But the new formation is on a much larger scale than the original bone, and there is much less finish and plan in its architecture. Gross provision is made for weight-bearing by the sclerosis of the new bone, which spreads early to some depth from the surface, and eventually extends to the greater part of the whole bone; but otherwise, the incidence and distribution of the alterations are somewhat erratic—e.g., areas of absorption may be contiguous to dense sclerosis, islets of very dense bone may occur in open porous cancellous tissue, and the central cavity may escape altogether or be considerably altered.\*

### CLINICAL SIGNS.

The striking clinical signs which follow upon the changes in the bone texture are: (1) *Thickening*; (2) *Elongation*; and (3) *Bending*.

1. *The Thickening* usually involves the whole bone, and the increase in size may be so great that the circumference may be double that of the normal

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\* Though the chief long bones in the upper and lower extremities—not omitting the clavicle—are those usually attacked, yet the metacarpal bones and the phalanges, and probably the corresponding bones in the foot, are not immune. It is, however, rare to find them obviously involved, and, even then, only some of them are affected.

bone. When many bones are affected some may appear to be only partially involved, but the disease gradually extends to the whole bone.

2. *Elongation* can be demonstrated by measurement when the corresponding bone on the other side is free from disease; but in the case of the leg or the forearm, if only one of the bones is affected it assumes a curved form which is obviously determined by an increase of length between two fixed points. In the leg it is usually the tibia which is at fault, and it acquires a marked anterior arch whilst the fibula remains straight. In the forearm, if the ulna is free, a diseased radius is apt to become conspicuously curved in a sigmoid fashion, placing the wrist and hand in the position of complete pronation, and deflecting them to the ulnar side.<sup>18</sup>

3. *Bending* is first seen, and is nearly always most noticeable, in the lower extremities. The curve in the tibia is an anterior one involving the whole bone, and the femur is usually arched forwards and outwards. The patient consequently acquires a bow-legged appearance, and owing to the bending of the necks of the femora there is some degree of coxa vara which adds to his clumsiness.

Should the bones of the upper extremity bend, the convexity in the humerus is usually anterior, but in the forearm there is more variety. The two bones may be bent backwards or forwards, and there is often some explanation suggested, such as the way in which a bedridden patient is accustomed to raise the body to the sitting position, lifting a heavy patient in nursing, wringing clothes in laundry work, and so on. The shape of the thorax is determined by the bending of the ribs in front of their angles, whilst changes in the shape of the true pelvis have been described (Wilks' case). In nearly all cases the influence of weight or force is evident, but elongation is a factor which under certain conditions has just been shown to be important.

*Formation of Deformities.*—It is clear that the deformities which characterize Paget's disease develop in the early stages when the bone is very vascular, and the new bone-formation, which is slowly replacing the original osseous structure, is widely porous, delicate, and insufficiently calcified. It is impossible to believe that the hard, dense, sclerosed bones, so many of which are preserved in museums, could yield under any strain they might be subjected to in ordinary circumstances, and clinical evidence supports this; there was little change in Paget's case in the last four years of life.

### HISTOLOGY.

The histology of osteitis deformans is very similar to that of osteitis fibrosa. The main features are the same. They are: (1) The disappearance of the original bone; (2) The substitution of a vascular connective tissue (osteogenic tissue) in place of it and its intertrabecular marrow; and (3) The formation of new bone from this connective tissue, in the first instance always by metaplasia.\* But there are several notable differences which give a

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\* The term 'metaplasia' is used here to distinguish bone-formation direct from the connective tissue from that which is associated with rows of osteoblasts.

peculiar and distinctive character to the microscopic appearances of osteitis deformans. These are so marked that it is possible to distinguish one disease from the other by the microscope alone.

1. The first to attract attention is the *regular and even distribution* of the new trabeculae throughout the diseased area. The whole of a section is occupied by the new osseous formations in various stages of growth, separated by comparatively narrow tracts of osteogenic tissue (Figs. 133, 134). In osteitis fibrosa, though trabeculae may be comparatively crowded in parts, there will almost certainly be large areas of connective or fibrous tissue in which the traces of ossification are absent or slight. This difference is least marked in the skull bones (flat bones and upper jaws), in which the histological characters and the great hyperostosis approach more nearly to the conditions seen in osteitis deformans.



FIG. 133.—Transverse section through a clavicle ( $\times 3$ ) showing the regular and even distribution of the bone trabeculae. From the same case as Fig. 134. The dark circumference to the section does not represent a compact cortical layer. (College Stores, R.C.S. Museum.)



FIG. 134.—Transverse section of a vertebra—from the same case as Fig. 133—to show the even distribution of the new bone formation. (Photographed by Dr. G. H. Rodman from a section of Dr. Greenfield's.) ( $\times 1.3$ .)

2. Equally conspicuous is the frequency with which the *trabeculae are seen to be decorated with internal curvilinear markings* (Fig. 135). Sometimes these clearly suggest a trabecula enclosed wholly or in part in a larger one, in other places they may show an outline waved and rounded like the edge of a cumulus cloud. This internal tracery is not peculiar to osteitis deformans. It may be seen, not infrequently, in osteitis fibrosa, but in that affection it is neither so

pronounced nor so universal. It points to bone-formation of a later date filling up spaces outlined by earlier trabeculae of the new formation, or fitting

into hollows caused by giant-cell resorption. By this mode of accretion fragments of the original bone may be interlocked with, or preserved in the

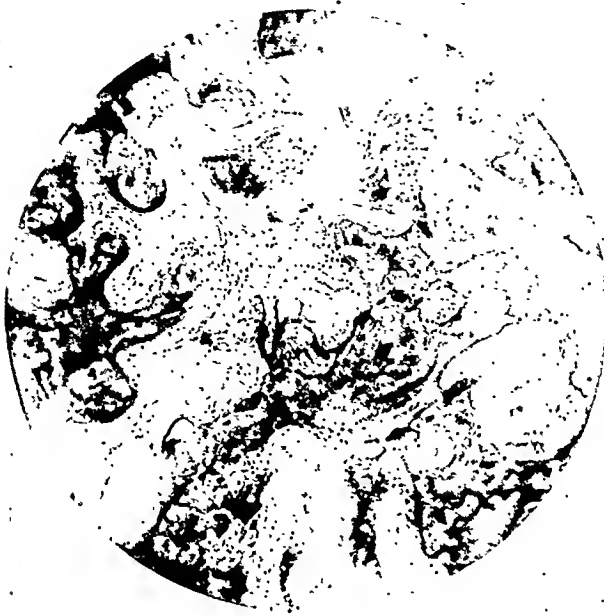


FIG. 135.—Trabeculae from a vertebral lamina (see case, p. 217), showing the curvilinear markings usually found in osteitis deformans. (Photographed by Dr. G. H. Rodman from a section of Dr. Greenfield's.) ( $\times 40$ .)

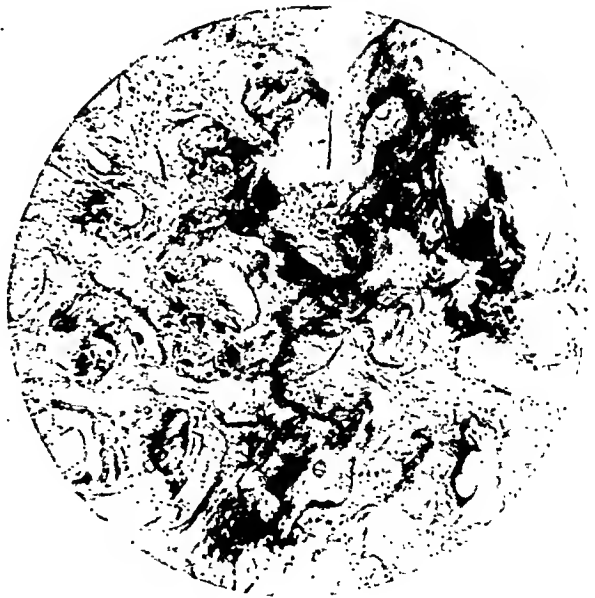


FIG. 136.—The curvilinear lines from a section taken from the skull of Dr. Hann's case. (Photographed by Dr. G. H. Rodman.) ( $\times 40$ .)

interior of, more recent material; or, again, the point to which the disease has extended, and possibly come to a halt, may be traced by a series of

scroll-like bays on the internal surface of the original compact laminae, filled in with more recently formed metaplastic bone. In some sections fine white lines mark part of the festooned outline, suggesting separation at the point of contact. These are probably artefacts produced in the preparation of the specimen.

Sometimes the fibrous groundwork of the trabeculae and the confused arrangements of its whorls are as conspicuous as in some cases of fibrous osteitis. The latter, and also the internal curvilinear markings, probably originate in the following way. A trabecula, forming in the first instance by metaplasia from the connective tissue, may assume such a shape that two parts of it may come into contact, fuse, and enclose a large lacunar space filled with osteogenic tissue. Similar lacunae may arise from the approxima-

tion and fusion of three or four trabeculae or ossific islets. Where so little space intervenes between the trabeculae as in osteitis deformans, it is difficult for one to increase much, especially if its growth is irregular, without coming in contact with some part of another. The formation of lacunar spaces of various shapes and sizes is therefore a necessary result of the plan of ossification. They are often spoken of as forming irregular Haversian systems—a description which is simple but confusing, for these spaces may become subdivided by the formation of

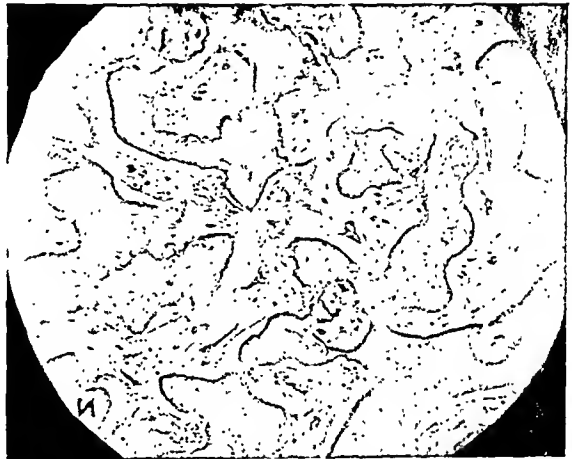


FIG. 137.—Section from the tibia shown in Fig. 139. The curvilinear lines are present, and the irregular plan of ossification from which they originate is well indicated. (Photographed by Dr. O. C. Gruner.)

smaller trabeculae in their interior, and the further progress of many is towards complete obliteration by osseous metaplasia of their contents (Figs. 135, 136, 137).

Absorption by osteoclasts is in some cases extraordinarily active—much more so than in osteitis fibrosa—though in that disease it is well marked. In osteitis deformans it may sometimes be seen in progress all over the section, and it is important to realize that the newly-formed bone is being steadily attacked.

Before obliteration of the lacunar spaces takes place their circumferences may be deeply eroded in places by osteoclasts, and so the bone that fills them up may acquire a festooned or bulging outline.

3. In osteitis fibrosa the complete disappearance of fat-cells is one of the distinctive features. In osteitis deformans fat-cells are not infrequently in evidence. In the former disease the appearance of a single fat-cell attracts attention; in the latter, they may occasionally be seen, scattered or in



groups, here and there among the trabeculæ. The conversion of fat-marrow is clearly less complete in osteitis deformans than in osteitis fibrosa, probably in consequence of the greater resistance opposed by the tissues to the toxic influence. This would explain why the central cavity usually persists and is much less liable to be completely obliterated over considerable areas than in osteitis fibrosa. No doubt most of the fat-cells seen in sections are

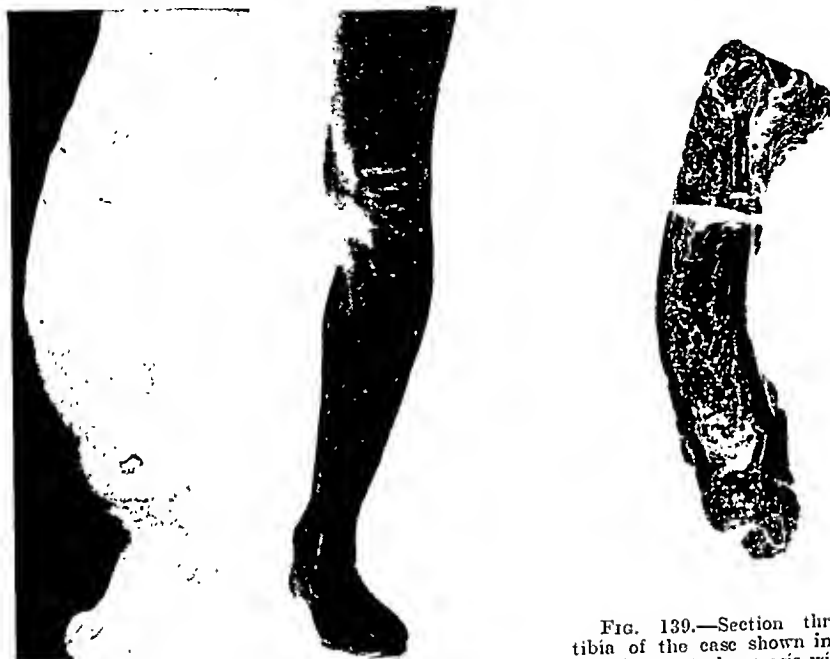


FIG. 138.—Photograph of Mr. Littlewood's 'single bone' case, with an ulcer which led to central necrosis.

FIG. 139.—Section through the tibia of the case shown in Fig. 138. Showing central necrosis with incomplete separation and an aperture opening on to the base of the ulcer. (Leeds Medical School Museum, old No. A322, new No. A817.)

unchanged portions of original fat-marrow, but occasionally a suspicion that some may have resulted from reversion of osteogenic tissue to fat appears to have some foundation.

4. Lastly, there is in osteitis deformans plenty of evidence of small-cell infiltration. But the cells are lymphocytes.

### 3. COMPLICATIONS.

**Fractures.**—Spontaneous fractures, apart from growth, have been recorded in several cases. Hector Mackenzie<sup>19</sup> described the case of a man, age 48, an in-patient at St. Thomas's Hospital, in whom the disease had existed eight years. He was thought to have fractured a rib when turning in bed. Shortly after his right leg seemed to 'give' half way between the knee and ankle, a lump rising on the front of the tibia at the place. Another instance was recorded by Stephen MacKenzie.<sup>20</sup> A woman whose tibia had been bending

for a year broke both bones of the leg by slightly knocking against a stool. She did not fall.

It is astonishing that similar fractures are not more frequently reported. On several occasions I have seen in skiagrams a line of fracture extending transversely some distance into a bone from the most prominent part of the bend—evidently a cleft resulting from strain upon the convex side of the softened bone. Such clefts, when there is no local tenderness, are probably filled in with fibrous tissue, though still shown by the X rays. One at least of the cases just quoted suggests a partial fracture, and partial fractures originating in this way would easily be made complete by a slight injury. Quite recently I saw an advanced case of osteitis deformans in a man of 60 who had had five fractures in various long bones from very trivial injuries during the course of the disease. The first (of a femur) had occurred when screwing up a powerful vice some twenty years before, and was apparently due to muscular action. The others occurred in the later years.

Ordinary fractures of bones affected by the disease unite as readily as those in normal bones, though good apposition is more difficult to obtain; and those who have had experience say that they do well after operative measures.

**Necrosis.**—In Sir Anthony Bowlby's case (p. 209, and *Figs.* 131, 131A) a small hard sequestrum lay in a cavity about the size of a nut. Similar cavities existed in other parts of the head, neck, and shaft, but were empty. Small masses of very dense bone sharply differentiated from the surrounding osseous tissue are of common occurrence in the skulls of these cases, and occasionally the appearances suggest the formation of a line of demarcation. The supervention of necrosis is best explained by the extreme condensation of the bone interfering with its circulation. Such an aseptic sequestrum may be gradually absorbed in the course of years, the cavities left being filled with granulation tissue, or possibly serous fluid.

Another variety was met with in the tibia in Littlewood's case (p. 209, and *Figs.* 138 and 139). A sinus led from an ulcer into the central portion of the lower end, and when the bone was bisected a partially separated green necrotic mass of rarefied cancellous bone—evidently of the new formation—was found in its interior. The ulcer had formed long after the disease had declared itself, and the septic osteomyelitis was evidently the result of infection from it. The track of infection was probably prepared by the substitution of open porous bone for the compact tissue, so that the connective tissue medulla would come into immediate continuity with the septic surface. This case emphasizes the importance of careful treatment of ulcers overlying bones affected by this disease. (Leeds Medical School Museum, No. A322, new No. A817.)

**Ulceration or Discoloration,** or both, of the tissues overlying the tibia, is not infrequent. It may be explained by impaired nutrition of the skin and superficial structures, which results from the changes in the bone and the retarding influence of muscular atrophy and diminished activity upon the venous return.

**Atheromatous Arteries** are almost a constant feature in these cases, and seen even when the disease occurs in early middle age. The arterial disease

does not interfere with an adequate blood-supply to the bones, for in the early stage this is excessive; nor is it likely that the atheroma is in any degree responsible for the bone changes, though this idea has been mooted. But it is quite possible that the deforming osteitis, the atheromatous changes in the vessels, and the gout, which these patients are said often to suffer from, may have a common exciting origin in toxins—possibly of intestinal or metabolic origin.

**Osteo-arthritic Changes** in the joints have occasionally been described (Lambert,<sup>21</sup> four cases). They do not appear to have been advanced, and when present were probably an accidental association. They are probably not more common than in other elderly folks, though the changed mechanics of the joints may sometimes favour their onset. Other accidental (?) troubles that have been recorded are lunacy (Beadles, Lambert), Huntington's chorea (Mackey), syringomyelia, and deafness (Jenkins).

**Malignant Disease.**—The frequency with which malignant disease supervened in his cases made a deep impression upon Paget, and the cases that were recorded soon after gave confirmation to the belief, that is even now widely held, that osteitis deformans usually, or at least very frequently, terminates by some malignant complication. And this view would gain support from the relative number of such specimens in museums. On the other hand, there is reason to believe that the frequency of this complication has been accidentally exaggerated. Packard<sup>22</sup> pointed out that in 66 cases there were only 8 of malignant disease, and 2 of non-malignant tumours. Gruner<sup>23</sup> states that up to 1902 only 14 of the recorded cases were associated with tumour formation (carcinoma 4, sarcoma 5, either carcinoma or sarcoma 4), whilst since 1902 (i.e. in more than one half of the reported cases) Higbee and Ellis<sup>24</sup> found mention of only 2 benign tumours. No doubt these figures do not represent the exact state of affairs, for many recorded cases were alive when described, and some malignant cases represented in museums have not been recorded. They afford, however, sufficient evidence to show that the first impression on this point must be readjusted.

When malignant disease does complicate osteitis deformans it is apt to appear more often than not in a bone which has not been obviously altered, or at any rate much altered, by the disease. Moreover, in a fair proportion of cases its connection with the bone disease is very doubtful (Wilks: epithelioma of internal surface of the dura. Goodhart: lymphomatous masses in liver, spleen, glands of mediastinum and abdomen; no primary source found. Robinson: sarcoma of cerebellum).

When it occurs in the bones it is usually in the form of an endosteal tumour; but if it arises in the cortical portion of the bone it projects prominently on one side and gives the impression of a periosteal origin (Univ. Coll. Hosp. Museum, No. 651A, Bone 64G).

#### 4. PATHOGENESIS.

It is interesting to note that osteitis deformans was very soon thought to be of toxic origin. Sir Jonathan Hutchinson<sup>25</sup> believed that it was "simply an infective osteitis, and that granting certain constitutional peculiarities it

was probable that the disease usually began as a consequence of contusion in one bone and spread by infection to others". Evidently he used the term 'infection' in the same sense as we should employ the word 'toxins', for further on he adds, "The theory, put concisely, is this, that the products of inflammatory action in any tissue are, when circulating in the blood, infective to that tissue throughout the body".

A different explanation of the toxæmia was suggested by Oetlinger and Lafont. They thought that the disease, which they met with in two brothers, whose father also had probably suffered, might be due to intoxication by mineral acids owing to the chlorine-laden atmosphere of the workroom in which they followed their occupation of laundrymen. The ingestion of hydrochloric acid would reduce the alkalinity of the blood, and set free indirectly a great quantity of lactic acid which would affect the evolution and structure of the bone. Their argument was supported by a table of the occupations of quite a number of cases in which the conditions were such that a similar explanation might be advanced.<sup>26</sup> At the present time this lactic acid theory cannot be seriously entertained.

Another theory was advanced by Gilles de la Tourette and Marinesco.<sup>27</sup> They found changes (atrophic, not true sclerosis) in the posterior median columns of the dorsal cord, and suggested that the bone troubles were trophic. Commenting on this idea, Packard writes, "The absence of all clinical evidence of trouble in the nervous system, and the results of many examinations which have shown the cord and bone nerves (von Recklinghausen) to be perfectly normal, render it unlikely that the lesions referred to have anything to do with the origin of the condition".

The possibility of the disease being due to micro-organic infection has, of course, been thought of. There is, however, a dearth of bacteriological investigations, and those that have been recorded are contradictory. J. C. Da Costa<sup>28</sup> writes, "Several Italian scientists claim to have found a diplococcus in the bone, and assert that they have found a similar diplococcus in the bone of osteomalacia. They prepared a vaccine and claim to have proved it serviceable. In two of my cases I removed a bit of bone from the tibia. Dr. Ellis made a careful study and found each piece of bone sterile. Beyond the statement of the Italian scientists there seems to be no evidence that bacteria can be causal". Levin<sup>29</sup> gives this additional information: "Da Costa, Funk, and others have tried to make a vaccine, but culture and animal inoculation experiments were negative". Morpurgo, Arehangelì, and Fiocea were no doubt the Italian investigators to whom Da Costa referred.

**The Toxic Theory Considered.**—Proof of the toxic theory is at present impossible, but the known facts are more in harmony with it than with any other: (1) There is an absence of any reliable evidence that the condition is due to micro-organic infection. (2) Adami,<sup>30</sup> in discussing the nature of chronic inflammation, points out that the process is long-continued and is accompanied by few or none of the cardinal symptoms of inflammation; that proliferative changes are more prominent than the vascular disturbances, and that the evidence of leucocytic migration is slight. Such changes when not of bacterial origin may be the result of repeated slight mechanical injuries or more often the effects of perverted metabolism and continued slight

intoxications affecting particularly certain tissues. The change in the medullary tissue in osteitis deformans conforms to this description, and there can be little doubt that it is brought about by toxic irritation. At the present time opinion certainly trends in this direction and attributes the origin of the toxins to tissue metabolism, or to the absorption of poisons of similar origin from the intestinal tract. Tubby<sup>31</sup> mentions a very suggestive case. It was that of a medical man who attributed improvement to the adoption of a diet rich in proteins and very sparing in carbohydrates, and the whole malady entirely to error in diet, viz., to lack of protein mainly, but partly to excess of starch. Eating potatoes never failed to produce a return of his pain.

In my Hunterian lecture on osteitis fibrosa I dwelt at some length upon a second important factor in its pathogenesis, viz., *the power to resist toxic influence inherent in the bones themselves*. That disease was assumed to depend upon an individual idiosyncrasy which permitted the bones to suffer damage from certain noxious blood-borne substances that produced no ill effects in ordinary people. The same assumption holds in the case of osteitis deformans. The toxins responsible for it can excite it only in the susceptible. On this hypothesis its sporadic incidence can be accounted for, and inherited tissue proclivity, which is probably the cause of the familial type, explained.\*

## 5. THE CORRELATION OF OSTEITIS DEFORMANS, OSTEITIS FIBROSA, AND OSTEOMALACIA.

When describing the histology of osteitis deformans I could not help concentrating attention upon the close resemblance that the microscopic appearances present to those of osteitis fibrosa. The essential characteristics were the same; any detectable differences were a matter of degree.

In considering the relations which exist between osteitis deformans, osteitis fibrosa, and osteomalacia, the pathological process, with which all three are intimately concerned, should be constantly borne in mind. That process, as I conceive it, begins by the entrance of certain toxins into the circulation. These toxins of metabolic or intestinal origin (?) are carried by the vessels to the bones and diffused by the lymph. The effect produced on the different constituents of the bone varies. The osseous framework, being a more vulnerable tissue than the marrow, has its vitality depressed beyond its power of recovery, but the connective-tissue basis of the marrow is excited to remarkable activity. The reaction which the toxic injury originates is shown by the striking alteration in the marrow. It is changed into an actively growing vascular connective tissue, whilst the fat vanishes from the cells, or the fat-cells themselves disappear. This new tissue plays a twofold part: (1) through its agency the damaged bone breaks up and disappears; (2) it becomes busily engaged in the manufacture of fresh bone by the metaplasia of its own fibrils and cells.

It should be noted that resorption, even of the new bone, is always going on, and sometimes is so widespread and active as to suggest a continuous

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\* There are three instances of a father and son becoming affected, and one of two brothers, two of a mother and daughter, and another of brother and sister. (Oetlinger and Lafont.<sup>26</sup>)

action of the toxins. At one time the reactionary soft tissue in the bone is probably largely in excess of the contemporary bony framework; but ossification, especially in osteitis deformans, outruns resorption. Howship's lacunæ, and lacunar spaces formed by the coalescence of trabeculæ, are speedily filled with new bone, and rows of osteoblasts are now seen behaving normally. In this way the bone may be reconstituted.

I would next point out certain peculiar features of this reaction in the case of osteitis deformans which seem to indicate that the bones in that disease are less seriously affected by the toxins than in osteitis fibrosa. These peculiarities are as follows: (1) the original bone is often not so completely removed as in osteitis fibrosa; (2) the reaction is apt to extend not so deeply into the fat marrow—the persistence of the central cavity in the long bones may be taken as an evidence of this; (3) it may be inferred from the regularity of the distribution of the new trabeculæ that the ossifying function is more vigorous, i.e., has been less depressed; (4) the interference with the strength of the bones is slower and less grave.

An explanation of the better defence against toxic influence put up by the bones in osteitis deformans may be found in their greater power of resistance. Vital resistance is here implied, but the greater strength and density of the mature adult bones as compared with those of childhood and adolescence must not be overlooked. Two clinical facts also throw light upon this matter. *The first* is concerned with the age at which the two diseases begin. Osteitis fibrosa is a disease of young people. It starts in the first or second decade of life. Osteitis deformans, on the other hand, is an affection of middle or old age. It rarely begins before the end of the 4th or the beginning of the 5th decade, and it may develop at any time between that and old age. *The second* is, that the subjects of osteitis deformans usually show marked evidence of arterial degeneration. The argument, therefore, leads to the inference that a *susceptible* individual with a sufficient reserve of resisting power may stave off osteitis fibrosa in youth, but succumb to osteitis deformans in old age when that resistance has been undermined by failing vitality and disuse.

This view of the close connection between osteitis deformans and osteitis fibrosa will help to clear up some of the confusion that exists with regard to them. In well-marked cases the diagnosis need never be in doubt; but there are cases—intermediary cases, perhaps—in which it is. I have already referred to a clavicle (No. A119b, Leeds Medical School Museum) supposed to be an example of osteitis deformans, and whose appearance does not belie that diagnosis. But microscopically it is osteitis fibrosa. The patient was a young woman 19 years of age, and *age will be found to be a very useful indication*. Radiologists, also, meet with and are puzzled by cases of undoubted osteitis deformans in which, in some parts of the skeleton, appearances are found which they are accustomed to associate with osteitis fibrosa. If the above explanation is sound, there is no inherent improbability of the occasional association in a single individual of lesions typical of both conditions.

But if osteitis fibrosa and osteitis deformans are to be looked upon as simply different expressions of the same disease determined by the dissimilar resisting powers of their victims, what then is the position of osteomalacia,

another toxic bone disease, which presents very different clinical as well as pathological phenomena?

Is there any connection between that affection and the other two? I believe there is, and will now give the reasons for my belief that the three conditions have a very similar—probably an identical—origin, and that, just as osteitis fibrosa or osteitis deformans is determined by some inborn property of the individual, so osteomalacia is similarly to be explained.

The clinical signs which are common to all three conditions—viz., softening, bending, and fracture of the bones—may be dismissed as of little importance in establishing this relationship. But if the histological features of the three diseases are contrasted, a very significant conception is suggested.

A description has just been given of the effect on the bone tissue of the toxic insult and of the reaction which it induces in the more recuperative marrow. In both osteitis fibrosa and osteitis deformans this reaction is very marked; the marrow is completely altered over large areas, and ossification in it is active. But in osteomalacia this reactionary change in the marrow is only developed to a slight extent, except under the stimulus of fracture or in the case of recovery taking place. In histological sections the dissolving and disintegrating trabeculae may be seen to be surrounded only by narrow zones of newly-formed connective tissue, and outside these zones tracts of ordinary fat marrow persist. There is no sign of ossification in this connective-tissue environment: bone disintegration alone is going on. (*Fig. 140.*)



FIG. 140.—Microscopic appearances in a section from an osteomalacic bone, showing trabeculae with decalcified borders, surrounded by proliferated connective tissue, but with normal medulla and fat persisting between the connective-tissue zones. There is no sign of active ossification. (*From a case of Mr. F. H. Mayo's, of Leeds. Photographed by Dr. O. C. Gruner.*)

These two facts seem to point to a degree of reaction so slight, that for the purpose of this argument reaction may be regarded as practically absent. In osteomalacia, then, it would seem that the tissues have not the power to react to toxic injury. Consequently, when the osseous framework disappears, the tissue replacing it, being possessed of too little vitality to maintain its own existence, or to advance to better things, peters out in fatty degeneration. Why should there be this failure to react? Surely the answer is to be found in the depressed and enfeebled condition of those in whom osteomalacia develops. Many who suffer from it have been subjected to debilitating influences such as poverty, privation, anxiety, rapid and repeated pregnancies, and residence in unhealthy dwellings and damp localities. This depressed vitality is one of the marked clinical features of osteomalacia.

It is important also to recognize that the course of osteomalacia is not infrequently punctuated by periods of apparent or partial recovery. If systematic examination of the bones of such a case were made, evidence

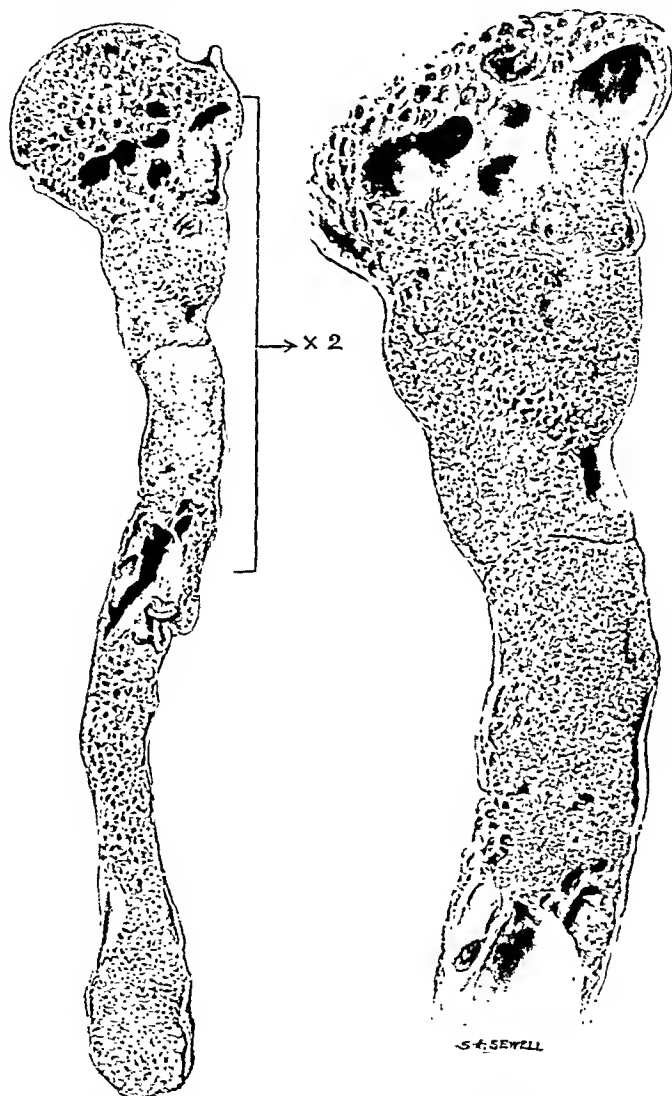


FIG. 141.—Drawing of the dried half of the humerus described in the text: [Goodwin's case. The picture on the right is a  $\times 2$  magnification of that part of the left figure included in the brackets. It shows the fine bony network which is made visible by the drying. In this case the part below the articular cartilage showed the microscopic appearances of osteomalacia, and a section cut from the surgical neck those of osteitis fibrosa. No. 735, R.C.S. Museum.]

of reaction would be found, and possibly osteomalacic changes and osteitis fibrosa could be demonstrated in the same skeleton or in the same bone. This combination has been found in one of Hunter's original preparations.



In the Royal College of Surgeons' Museum are the two halves of a humerus which has been divided vertically, Nos. 734 and 735. One half has been mounted as a wet specimen, the other as a dried one (not macerated).

The wet specimen, No. 734, "the subject of mollities ossium", shows a fracture at its lower end. The bone is of natural shape, but except for a very thin and rarefied cortex, and for a small amount of cancellous tissue adjacent to the articular cartilages, it is composed of a pale, soft structure which feels fibrous and gritty.

The dry specimen, No. 735 (*Fig. 141*), is "shrivelled into a tough flexible mass like a piece of dried muscle". Its interior is filled from side to side and from end to end—except where it is interrupted by a cystic space—with a very fine reticulated osseous tissue.

Professor Shattock picked a spicule from the cancellous tissue just below the upper articular surface of No. 734, and carefully prepared it without artificial decalcification. It showed the characteristic histological appearances of osteomalacia, viz., a typical double contour to the trabeculae due to a calcified central portion surrounded by a decalcified border; also a granular appearance of the central calcified portion, and the lacunae surrounded by a pale area with innumerable lines radiating laterally.

He next prepared a longitudinal section cut from the surgical neck (734), including the periosteum and a considerable depth of the gritty fibrous interior, and stained it with eosin and hæmatoxylin. The chief portion of this specimen was typical of osteitis fibrosa. A regular network of newly-formed trabeculae spread through a continuous field of connective tissue. The trabeculae were only partially calcified, the lime salts being deposited irregularly in an uncalcified matrix, and they were being formed from the connective tissue without the mediation of layers of osteoblasts. The subperiosteal cortical layers were long and atrophic, without any decalcified bordering, but the spaces between them were filled with fat—a condition not inconsistent with an interrupted osteomalacic process.

The case was published by Goodwin,<sup>32</sup> and Hunter gave an account of the dissection of the arm which had been sent to him.<sup>33</sup>

The patient was a woman, age 34. After suffering from severe rheumatic pains in the limbs, she broke her leg tripping against a brick. She soon after became pregnant, and broke her left femur getting out of bed. She was safely delivered, but suffered several more fractures, and again becoming pregnant she gave birth to a living child. Subsequently her health improved, but during another pregnancy (the tenth) the fractured bones, which had united, broke again, the dissolution of the callus being preceded by excruciating pain. Several new fractures then occurred, and on her death after three years' illness the bones were found to be so soft that they could easily be cut with a penknife. The cranium and vertebrae were much affected, and the bones of the lower extremities were the least diseased.

From an observation of Ziegler's in connection with the description of osteomalacia, that "new osteoid tissue is not infrequently found in bones which have not perceptibly bent or yielded",<sup>34</sup> it is evident that similar facts have been recognized before; and from such facts and from the structure of callus in these cases the conclusion naturally follows, that when recovery does take place in osteomalacia the bone changes characteristic of that condition give place to those of osteitis fibrosa.

The argument may be summarized as follows :—

1. Certain diseases of the bones owe their origin to the action of toxins—probably those of metabolic or intestinal origin.

2. Predisposing to their development an idiosyncratic susceptibility on the part of those who suffer from them may be assumed.

3. The ability of the bones to resist toxic influence is a factor of great importance.

4. The relative intensity of that resistance can be roughly gauged by the extent and character of the reaction excited in the soft tissues of the affected bones.

5. If the vitality is good, the resistance opposed to the toxins may ward off harmful effects till the constitutional strength is reduced by age and disease. The reaction under these circumstances is strong, and the resulting affection takes the form of *osteitis deformans*.

6. On the other hand, if vitality is feeble the disease appears earlier—in childhood, adolescence, or early manhood. The reaction is fair, but not so vigorous as in osteitis deformans. The form the disease then assumes is that of *osteitis fibrosa*.

7. Lastly, when owing to the severe depression of the patient's vitality, usually by untoward circumstances, the power to react is absent *osteomalacia* develops.

## 6. CAUSES THAT MAY DETERMINE THE ONSET OF THE DISEASE.

The onset of osteitis deformans appears with some frequency to be determined by an accidental circumstance or condition which no doubt causes some local or general depression of vitality. Injury is the most important of these predisposing causes. It is said to be particularly frequent in the single bone cases (*see* p. 209), but it is often mentioned in the records of cases of generalized disease. Inflammation of the skin over both legs was suspected in one case. It lasted four or five months, and left large pigmented areas when it subsided. Five years later the first signs of the disease appeared in the legs.<sup>25</sup>

The following history was given by a man, age 58, under the care of my colleague, Mr. J. A. Coupland, at the Leeds General Infirmary, with an enlarged bent tibia—the only bone affected. Twenty years ago he sprained his knee. When 40 years of age he sustained a fracture (?) just above the ankle, which he treated himself. Eight years ago he suffered with blood poisoning from a wound caused by a rusty wire. The leg was enormously swollen, and marks of more than a dozen incisions were present. The leg was noticed to be bending from that time, and more so in the last five years. (From notes by Mr. A. W. Birtwistle.)

Overfatigue, fatigue from long nursing, injury with attendant fright, frequently getting wet through, have all been thought to exert a predisposing influence in particular cases; and, indeed, any debilitating illness, whether of micro-organic origin or not, might act in this way.

A history of syphilis has been present in a few cases (Paget<sup>36</sup>); but it

is rare for this to be obtained or for any sign of that disease to be found. Da Costa<sup>37</sup> states with reference to the Wassermann tests that "four-fifths of the cases have a negative reaction". "A consistent positive reaction has never been found in an uncomplicated case".

**Internal Glandular Secretions.**—As these secretions are undoubtedly associated with certain bone conditions, it is natural that cases of osteitis deformans should be carefully scrutinized for any evidence of their influence. The facts that have been gleaned have been considered by Hurwitz, and by Higbee and Ellis, and the opinion of the former,<sup>38</sup> which appears to be well founded, is "that the assumption that there is a causal connection between the internal secretions and osteitis deformans is mere speculation".

**The Nature of the Morbid Process.**—From the beginning it has been regarded as inflammatory. Paget and Butlin discussed this point at length in the original paper, and were decidedly of that opinion. Adami and MacCrae<sup>39</sup> find no evidence of the inflammatory stage and regard the process as metaplastic. This has reference to the alteration of the medulla from fatty or abundantly cellular to fibroid, but hardly coincides with Adami's observation on chronic inflammation which I have summarized in discussing the toxic theory. This question was dealt with in my lecture on osteitis fibrosa, and the same reasons that were held to indicate that that affection was inflammatory apply in the case of osteitis deformans.

I wish to acknowledge the debt of gratitude that I owe to the late Professor Shattock in connection with this lecture. Always kind, sympathetic, and approachable, it might almost be said that it was prepared under his supervision. My thanks are also due to the curators of the different museums from which I have drawn my material, and to others, for their kind help and courtesy, and to Dr. G. H. Rodman for the pains he has taken to bring out in the microphotographs the points which I wanted to make, and to Messrs. George, Steward, and Wilson, of the Royal College of Surgeons staff, for their valuable help, which is evidenced by the illustrations. Nor must I forget the kindness of Dr. O. C. Gruner, though it is many years since he took the plates which I have now made use of.

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## A CONSIDERATION OF TWO CASES OF CYSTADENOMA OF THE PANCREAS, AND THEIR PROBABLE RELATIONSHIP TO POLYCYSTIC CONDITIONS FOUND IN OTHER VISCERA.\*

BY E. ROCK CARLING AND J. A. BRAXTON HICKS, LONDON.

### CLINICAL NOTES.

THE first case, W. C., a male, age 53, a patient of Dr. C. H. Pring, had been treated for hæmoptysis in 1922. No tubercle bacilli found in the sputum. In September, 1924, feeling 'out of sorts' but having no definite symptoms, he consulted Dr. Pring, who found glycosuria. On a restricted diet the sugar quickly disappeared. Seen in November by one of us (E. R. C.) with Mr. Arthur Evans, he complained of nausea and abdominal pain. He had not

lost flesh. In the epigastrium, mainly to the right of the middle line, was a hard tumour of rounded outline, dull on percussion, apparently separable from the liver. No jaundice, and stools of normal appearance. Mr. Evans thought the lump pancreatic, but there was also some question whether it was not a malignant growth of the stomach or transverse colon.

Exploratory laparotomy (E. R. C.) revealed a cystic tumour between stomach and liver, about 4 in. by 4 in. by 2½ in. It was found that, when freed from all other connections, the tumour arose by a narrow pedicle from the upper border of the head of the




FIG. 142.—Cystadenoma of the pancreas removed in Case 1. Situated behind the lesser sac, between stomach and liver, the actual attachment to the pancreas was by a pedicle of not more than ½ in. wide; the pancreatic tissue exposed was slightly spongy in texture.

pancreas, near the mid-plane of the body. (Figs. 142, 143.) After removal a drain was left in, and from a purely surgical point of view there was no trouble. Pneumonia set in, and very severe glycosuria. The hyper-

\* From the Laboratories of the Westminster Hospital.

glycæmia fluctuated violently and was very imperfectly controlled by insulin, despite repeated estimations of the sugar content of the blood. Death supervened on the tenth day.

The second case was that of F. B., male, age 76, under the care of Mr. William Turner, to whom we are indebted for permission to publish these notes. Admitted April 24, 1922. For eighteen months he had been aware of a lump in the abdomen, and had been losing flesh. Three months before admission he had had an attack of severe abdominal pain, during which, for a fortnight, he was very ill. Since that date he had had increasing constipation.

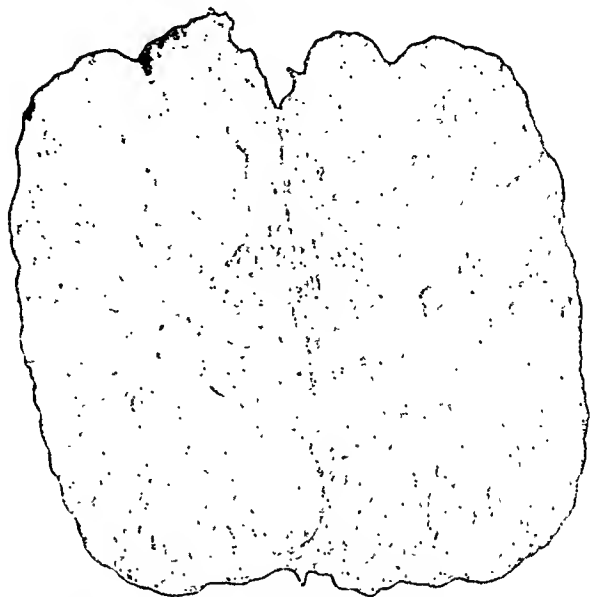


FIG. 143.—Sectional view of the tumour illustrated in Fig. 142.

Temperature, pulse, and respiration were normal.



FIG. 144.—Post-mortem specimen. Pancreas containing cystadenoma; from the case of F. B., under the care of Mr. William Turner. (*P.M.* 30-213.)

A large hard tumour occupied the right hypochondrium, extending into

the right lumbar and epigastric zones. Its lower margin could be defined, and projecting backwards from it were two rounded masses the size of oranges. The diagnosis was "probably metastases in the liver, from carcinoma of the hepatic colon", but in view of the recent acute attack of pain, etc., the possibility of an inflammatory mass from some lesion of the gall-bladder or in its neighbourhood was also considered.

Laparotomy revealed a cystic mass in and below the pancreas, the cysts containing a glairy fluid. A mass of adhesions in the upper part, binding down the colon to the liver, was regarded as the result of peritonitis from rupture of one of the cysts, at the time of the acute illness.

The patient died seventeen days after the operation, and at the necropsy the ovoid tumour illustrated in *Fig. 144*, 6 in. by 4 in., was found replacing



FIG. 145.—Section from the tumour in *Fig. 142*, showing the low cuboidal cells lining the cyst spaces. Where these cells can be seen free they have a polyhedral form, ample clear protoplasm, and a large deeply-staining single nucleus. The ground substance of the tumour is connective tissue, in places cellular, in others well-formed fibrous tissue. (C.R. 21-882; Section No. 4109A.)



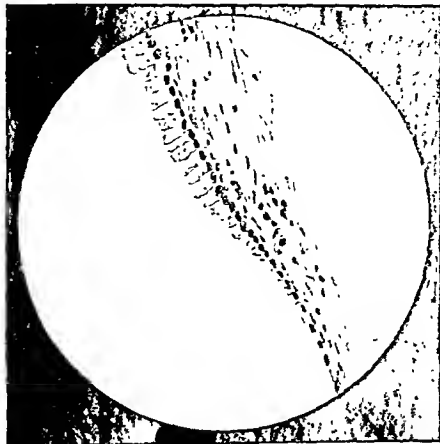
FIG. 146.—Section of tumour shown in *Fig. 144*. As contrasted with *Fig. 145* it will be seen that the ground substance is scanty, but that the general arrangement, and the type of cell lining the cysts, are closely similar. (P.M. 30-213. Section No. 3679A.)

the head of the pancreas. The body and tail were normal, though the duct, which passed right through the mass freely into the duodenum, was dilated. The central portion of the tumour was calcareous, and the whole weighed 36 oz. It was adherent to and pressed on the colon, but there was no evidence of 'infiltration' or of secondary deposits.

Microscopical examination of these two tumours (*Figs. 145, 146*) reveals a striking similarity between them, so much so that one can at the outset assume that the pathological conditions are the same in each case. There are, it is true, individual peculiarities, but these are rather in degree of tissue changes than in actual type of tumour. Thus the basic appearances seen are those of a coarse network of fibrous or fibromuscular trabeculae enclosing spaces lined with cells of polyhedral, low cuboidal or flattened types. There is no

suggestion of 'malignancy' in the accepted histological sense; there is not the slightest resemblance in the structure of these tumours to the normal or even malignant pancreatic cell arrangement; and what seems to us very

FIG. 147.—Part of the wall of a 'solitary' cyst enucleated partly from the upper aspect of the head of the pancreas, partly from the wall of the pyloric portion of the stomach. The wall is like that of the other cysts; the lining cells, columnar at one part, become low cuboidal, and eventually flattened, as the wall becomes thinner. This particular area of the section is, of course, specially chosen as illustrating the changes columnar epithelium undergoes either under pressure or in association with stretching of the connective-tissue wall. (*C.H.*; *Clin. Rep.* 20-1116; *Section No.* 3884A.)



important also, no connection with the pancreatic ducts. The last factor was partly exemplified by the ease with which a probe passed through the pancreatic duct into the duodenum, through the whole of the main mass of



FIG. 148.—Liver of A. W., a patient of Mr. W. G. Spencer's. Weight 167 oz. Polycystic disease of the liver and kidneys. The case was admitted as an acute abdominal emergency: severe pain, great distention, complete constipation. The post-mortem report states that death was due to hepatic and renal inadequacy. The kidneys, right 36 oz., left 39 oz., were typical examples of polycystic disease. (*Sections* 1264A to 1265A; *P.M.* 27-42.)

the tumour, in the case F.B. In the same case there was a suggestion that these cystic spaces might have arisen as lymphatic spaces of new formation, but a consideration of the whole tumour (or of both tumours) in



our opinion negatives such a suggestion; indeed, the only difference between the pancreatic tumour in the two cases is that the cystic spaces in the first are larger and more irregular in size and shape than are those of the second, in which the texture is much more like a close-meshed sponge.

It is interesting to note that in a solitary cyst which was embedded partly in the pancreatic substance and partly in the muscular wall of the stomach, and removed by one of us (E. R. C.), similar appearances can be found. There is a wall of fibromuscular tissue; the lining cells vary from tall columnar with a well-marked deeply-staining nucleus to partly flattened cells. Further, in one part (*Fig. 147*) the transformation from the one type to the other, passing through a low cuboidal cell stage, can be seen, and this change is found where the thick fibromuscular wall becomes attenuated. Moreover, even this 'solitary cyst' contains in some parts of its wall smaller cysts of precisely the same structure as the main cyst.



FIG. 149.—From the liver illustrated in *Fig. 148*. The similarities between this and the section in *Fig. 150* do not need particularization. (*P.M.* 27-42; *Section No.* 1264A.)



FIG. 150.—From the kidney of the same case as *Fig. 149*. On the upper edge of the drawing the lining cells of a cyst of medium size. Many have apparently double nuclei, but they are otherwise similar to those in the other cysts. (*P.M.* 27-42; *Section No.* 1267A.)

In our investigation of these three tumours, we were led by their very similar appearances to reconsider the structure of such examples as the one illustrated showing polycystic disease of the liver and kidneys (*Fig. 148*), and a similar one, formerly under the care of the late Mr. Charles Stonham, in the Museum of the Hospital (No. 563, in Museum Catalogue), in which also both liver and kidneys were involved, the liver to a very much less degree than our illustrated case; and still further to consider their probable relationship to a case of cystadenoma of the bile-ducts published from this laboratory by Mr. Arthur Evans.<sup>1</sup> In the case of polycystic disease of the liver, though the connective-tissue walls (or, perhaps more correctly, the connective-tissue masses in which the cysts are embedded) are very much more cellular, and much less definitely fibrous in type, than those of the pancreatic cases, yet the

cells lining the cystic spaces are of closely similar appearance to those seen in the pancreatic tumours, and indeed do not differ materially from those seen lining the spaces in the kidneys of the same case; it would be possible to place under the field of the microscope a part of any one of these cysts, save possibly the renal, and mistake it for any other. This has become very apparent to one of us (E. R. C.) during the process of drawing the appearances for the illustrations reproduced (*Figs. 149, 150*).

In the case of the cystadenoma of the bile-ducts referred to, the cells lining the ducts are of a very tall columnar type, with nuclei elongated in the axis of the cell (*Fig. 151*). They do certainly suggest an origin from bile-ducts, but the tissue in which the cysts are embedded is not liver parenchyma, but of a fibrous nature; nor have we ever established any connection between the cysts and existing bile-ducts. Further, these cysts did not contain bile. The patient, after the first operation, did not make any great progress towards recovery; the liver became irregularly enlarged, and was of enormous size when the patient died. Unfortunately permission for a post-mortem was refused, and we are indebted to Mr. Arthur Evans for these further notes on his case. It is our belief that the condition of cystadenoma of the bile-ducts, where such localized conditions have so been described, is merely an early and localized condition of the more advanced polycystic disease of the liver, and that probably Mr. Arthur Evans' case terminated in this way.

It is of interest to note that the patient from whom the 'solitary cyst' was removed had also another abnormality, in that the left kidney was shaped like two-thirds of a horse-shoe, and the ureter crossed the lower median pole, which extended beyond the middle line. This indicates some aberration in the differentiation of the Wolffian body, and will be seen to have some bearing on our final conclusions.

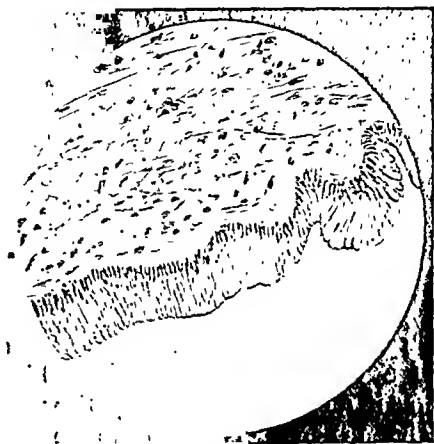


FIG. 151.—From Mr. Arthur Evans' case of cystadenoma of the bile-ducts. (*Brit. Jour. Surg.*, 1921, ix, 155. C.R. 17-476: Section No. 2305A.) No trace of liver substance is to be found in the sections: the walls of the cyst are rather more cellular than those of the others illustrated, but in parts the fibrous tissue is well formed. The cells are high narrow columnar, and the nuclei elongated in the same axis: but in many of the cysts of the other cases, columnar cells of mucus-secreting type are to be found, even when the bulk of them are, or have become, cuboidal or flat. Without questioning the origin from biliary duct cells, it is open to doubt whether the cystadenoma is derived from the fully-developed and functioning ducts of the adult liver to which the tumour was attached.

#### BRIEF NOTES ON OTHER RECORDED CASES OF POLYCYSTIC DISEASE OF THE PANCREAS, ETC.

Polycystic disease of the kidney has been observed in at least one case of cystadenoma of the pancreas, and since looking up the bibliography of

similar cases we have come across the figure illustrated in our *Fig. 152*, which is from a case of polycystic 'degeneration' of the spleen, of which condition a good example also is illustrated in Silvestrini's *Patologia e Chirurgia della Milza* (*Fig. 153*). The histological appearances could be matched exactly in any of our sections.



FIG. 152.—Section from a case of polycystic disease of the spleen. (Reproduced by the courtesy of Dr. E. H. Pool, from the *Surgery of the Spleen* by Eugene H. Pool and Ralph G. Stillman: Appleton, 1923.)

those in the tail, the largest the size of a cherry-stone. The right kidney was in a condition of polycystic disease. The pancreatic tumour was reached through the lesser sac, as was ours; but unlike our *Case 1*, was "easily delivered". Clinically there was great emaciation, and the diagnosis was "cancer of the pylorus, with hepatic metastases". The functions of the pancreas were normal. Recovery occurred after the operation. The cells lining the cysts were of a columnar type. Malcolm's<sup>4</sup> first case was a female, age 50, who complained of loss of flesh, debility, and mæna. The urine was normal. A mobile tumour, 4 in. by 4 in., was situated in the left upper abdomen, and the clinical resemblance was to a malignant growth in the left kidney. In this case "some of the proper glandular tissue suggested that of the pancreas". "In some places there were masses of polyhedral cells, some of striking size

Practically the whole recent literature is to be found in Gross and Guleke's "*Die Erkrankungen des Pankreas*", 1924,<sup>2</sup> where there is a short account of cystadenomata, but no plate of microscopical appearances. One or two additional cases may be noticed briefly.

In Bolt's case,<sup>3</sup> in a female of 51 years, there was involvement of the whole pancreas, the cysts in the body and head being larger than

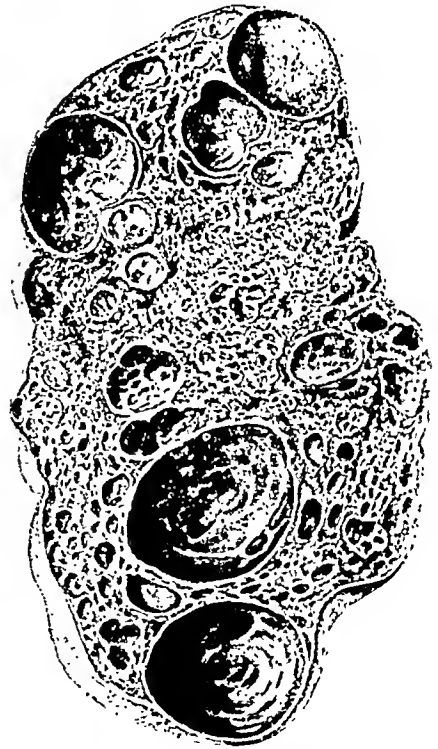


FIG. 153.—Polycystic spleen in section. (Reproduced by the courtesy of Professor Silvestrini, of Rimini, from *Patologia e Chirurgia della Milza*: Capelli, Bologna, 1924.)

some of striking size

and with more than a single nucleus." Malcolm's second case was a female of 49, in whom the tumour was thought to be a hydronephrosis of the left kidney. The cysts (two large and several smaller) contained blood, and a fluid that was amylolytic, but in this case there had clearly been rupture and access of pancreatic juice. Pitchford's case was easily shelled out, though it weighed over 2 lb., but the fluid showed no trace of pancreatic ferments. The cells present were polyhedral, with clear body and deeply-staining nuclei. The cysts were lined with short columnar cells.

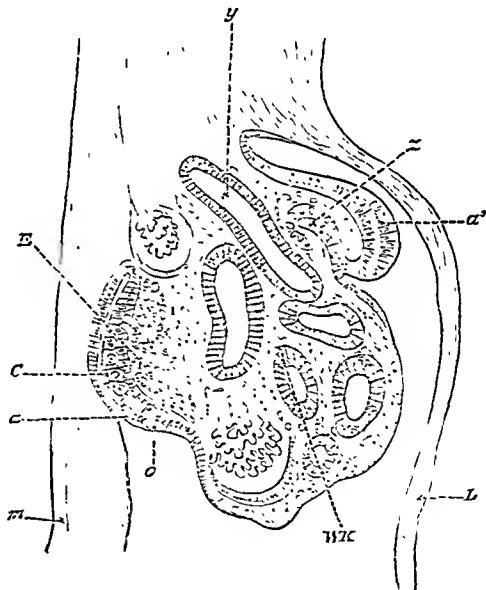
### CONCLUSIONS.

It seems to us that at least the more isolated of these cystic masses are embedded in the organ 'by accident', their cellular characteristics, whilst adenomatous in type, differing quite markedly from those of the organ in which they are found. Though it must be confessed we have not tested the ferment activity of the fluids in our cases, yet the cyst contents of the

FIG. 154.—Section of the Wolffian body, developing pronephros, and genital gland of the fourth day. ( $\times 160$ . After Waldeyer.)

*m*, Mesentery; *L*, Somatopleure; *a'*, Portion of the germinal epithelium from which the involution (*z*) to form the pronephros (anterior part of Müllerian duct) takes place; *a*, Thickened portion of the germinal epithelium in which the primitive germinal cells *C* and *o* are lying; *E*, Modified mesoblast which will form the stroma of the ovary; *WK*, Wolffian body; *y*, Wolffian duct.

(Reproduced by permission of Messrs. Macmillan & Co. Ltd., from Balfour's *Comparative Embryology*, Vol. II.)



pancreatic cases were of the same mucinous type as those we noted in the polycystic kidneys and in the liver, and there is no reason to suppose that they had any particular ferment action. We believe that it is not going too far to say that all these conditions can be brought into line and explained by assuming the inclusion of isolated portions of the Wolffian body either in the enteric buds forming the liver and pancreas in the embryonic spleen as it develops in the ventral mesogastrium, or of that part of the intermediate cell mass in which the kidney forms.

In the embryo these organs all arise in the immediate vicinity of the Wolffian body, and although it has been 'fashionable' for a long time to

dispute this theory of the origin of the polycystic condition of the kidneys, yet no one has really advanced a better one or one that will cover the whole ground, and it is in our opinion a reasonable suggestion that on this theory we can also explain these interesting and rare polycystic conditions of the pancreas, liver, and spleen. A reference to *Fig. 154*, reproduced from the diagram by Waldeyer given in *Quain*, 10th edition, vol. i, plate 1, p. 120, showing the embryo of the chick, gives, for example, the type of cell and structure found in these tumours.

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## REFERENCES.

- <sup>1</sup> EVANS, ARTHUR, *Brit. Jour. Surg.*, 1921, ix, 155.
- <sup>2</sup> GROSS and GULEKE, "Die Erkrankungen des Pankreas", *Enzyklopaedic der klin. Med.*, 254 (extensive bibliography).
- <sup>3</sup> BOLT, R. F., *Ibid.*, 1913, i, 142.
- <sup>4</sup> MALCOLM, *Clin. Soc. Trans.*, xxxix, 175 (short bibliography).

## THE PATHOLOGY OF HYDRONEPHROSIS.

By H. P. WINSBURY WHITE, LONDON.

(Being a Hunterian Lecture delivered at the Royal College of Surgeons of England on February 4, 1925.)

IN spite of the voluminous literature which exists on the subject of hydronephrosis, there is still a widespread diversity of opinion as to the primary etiological factor in the pelvic type of the disease. This has been largely due to lack of opportunity in the past to study the earliest examples of the condition; and because investigations have been generally carried out on specimens in advanced stages of dilatation, cause and effect have been largely confused.

To hydronephrosis from such undisputed causes as calculus or growth involving any part of the urinary tract, or from any form of obstruction to the lowest urinary passage, I shall make no further reference, except to point out that the striking feature in the pathological anatomy when due to any of these causes is that the dilatation advances more markedly in the calices than in the pelvis. *Fig. 155* illustrates this point. It is an advanced example of calculous pyonephrosis resulting from the small stone in the renal pelvis.



FIG. 155.—Renal type of hydronephrosis, resulting from stone in the pelvis of the kidney. (Lantern slide.)

When the obstruction is due primarily to a stenosis in the region of the pelvo-ureteral junction, the dilatation is more marked in the pelvis than in the calices. Hence the term 'pelvic hydronephrosis'. The result of this pelvic distension is an abrupt separation of pelvis from ureter. This definite demarcation between these two components of the efferent system is essentially a pathological one, for it is not possible in a healthy kidney to indicate accurately, either macroscopically or microscopically, the actual locality where the pelvis ends and the ureter begins.

I have been able to note details of 159 cases of hydronephrosis. A large number of these were from the literature, others were from the records of the Royal Free Hospital and from notes of my own personal cases. I have arranged these in *Table I* according to the chief features of their pathological anatomy as follows:—

*Table I.*—CLASSIFICATION OF 159 CASES OF HYDRONEPHROSIS  
ACCORDING TO CERTAIN PATHOLOGICAL FEATURES.

				NO. OF CASES
Pelvic hydronephrosis—				
Simple unilateral	..	..	..	121
Simple bilateral	..	..	..	2
Ectopic kidney—				
Unilateral	..	..	..	5
Horseshoe kidney—				
Unilateral	..	..	..	5
Bilateral	..	..	..	1
Double ureters—				
Unilateral	..	..	..	1
Bilateral	..	..	..	1
Dilated bladder, both ureters, and kidneys—				
Without apparent cause	..	..	..	3
Phimosis	..	..	..	1
Atresia of external urinary meatus				1
Dilatation of whole ureter and kidney—				
Unilateral	..	..	..	6
Bilateral	..	..	..	8
Dilatation of upper half of ureter and kidney—				
Unilateral	..	..	..	2
Traumatic hydronephrosis—				
Injury to ureter (verified)	..	..	..	2
Total				159

I have not discriminated between those in which there is a blood-vessel which is said to be compressing the ureter and those in which this is not apparent, for the reason that after division of the offending vessel the appearance of the two varieties does not differ, and there is no reason to believe that the etiology in the two cases is not the same. In each case it is a hydronephrosis with the dilatation beginning at the pelvo-ureteric junction. *Table I* shows that 85 per cent of the 159 cases were of this variety, and that more than 97 per cent of these were unilateral.

### CONGENITAL HYDRONEPHROSIS AND HYDRONEPHROSIS IN CHILDREN.

The term 'congenital' has been freely used to describe not only those cases which occur in foetal and in the early periods of independent existence, but also many cases which first attract attention at some period of adult life. Most of the earliest cases may be definitely accepted as congenital, for many are found in association with abnormalities of this nature involving other parts of the body, such as spina bifida and imperforate anus. Blackwood, in writing of 28 cases that he collected of hydronephrosis in infants, states that he found an associated congenital defect in 9. It is of some interest to note that heredity also plays a part, for the same writer refers to the reported case of a woman who gave birth to three premature children, each with a hydronephrosis.

Unlike the cases first noted in adult life, hydronephrosis in children is frequently accompanied by dilatation of the corresponding ureter. Of 32 cases in children under twelve years of age that I have noted from the literature, 17 had dilatation of one or both ureters, both being involved in 10 cases. In the remaining 15 cases the dilatation began at the pelvo-ureteric junction. The length of life when both ureters are dilated is generally short, most of the cases dying within the first two years of life. This fact was noted by Davis, who refers to a report of 25 cases of bilateral dilatation, nearly all of which were under ten years old.

With regard to the cause of the dilatation, in two only of the bilateral cases that I have collected was a definite obstruction to the outflow of urine from the penis described. In one instance this was a phimosis, in the other a narrow external urinary meatus. In a few cases constrictions at the vesical ends of both ureters were described as the cause. Several instances were met with in which the bladder as well as the ureters was dilated without apparent reason. It is important to recognize that dilatation of the bladder may be present when no urethral obstruction can be found. Bard has described 3 cases, all in males, at the ages of 16, 36, and 48 respectively. In several cases of unilateral dilatation of the ureter, a stricture at the junction of the ureter with the bladder was described. In others the cause was not indicated. It would be interesting to know whether dilatation of the bladder ever accompanies unilateral ureteric dilatation as a congenital condition. Poynton described the case of a boy, age 6, in whom the dilated left ureter was demonstrated in a cysto-ureterogram, but it was not ascertained whether the other ureter was not dilated to a lesser degree. Unilateral dilatation may escape recognition till puberty or later.

When the ureter is not involved in the dilatation there is always to be seen an abrupt demarcation which separates a distended pelvis from a narrow ureter. The impression from this is generally that the size of the ureteral lumen is inadequate as an efferent urinary duct. In some of the most striking foetal and infantile cases described no lumen of the ureter existed at all.

The question of inadequacy of the ureter is one of very great interest, and was brought forward many years ago by Bland-Sutton as the chief cause of the type of hydronephrosis which begins at the pelvo-ureteric junction. That the condition occurs also in an acquired form as the result of chronic inflammation is certain from the evidence I have brought forward. Geraghty and Frontz in 1918 expressed the view that abnormalities in ureteral calibre are probably the most important factors in the production of hydronephrosis. When one recalls that cases occur which first give rise to symptoms during any year of childhood, adolescence, or adult life, it becomes extremely difficult to know where to draw the line between the congenital and the acquired disease. But the fact that the great majority of cases manifest their first signs of disease after the age of twenty speaks strongly in favour of these being acquired rather than congenital.

Such features as high or oblique insertion of the ureter into the pelvis, and contracted or valve-like pelvic outlet, often referred to as congenital, are seen only in advanced stages of hydronephrosis, and are a consequence



out the age incidence expressed in decades, of 50 cases shows that 23 of these manifested their first symptoms between the ages of 20 and 30. It is to be noted that the onset in one case was at the age of 75. From this it appears that hydronephrosis is a disease which may occur at any period of life.

In a further study of the same cases 33 were found in females and 17 in males. The left kidney was the seat of the disease in 26 instances, and the right in 24. There was no case of bilateral disease. Other writers have reported that hydronephrosis is twice as common in women as in men.

REFERENCES.—9, 15, 21, 25, 26, 29, 33, 36, 42, 43, 44, 46, 47, 50, 53, 54, 58, 59, 67, 68, 74, 76, 79, 81, 82, 86, 91, 100, 106, 107, 108, 109, 116, 117, 119, 123, 124, 125, 128, 130, 131, 132, 133.

### INFLAMMATION AS A CAUSE OF HYDRONEPHROSIS.

From the point of view of establishing the primary etiological factor of hydronephrosis, it is on the earliest cases that observations may be considered of greatest importance. It is, however, only as a result of urteric catheterization and pyelography that one is able to detect with certainty, and thus

has an opportunity to study, the disease in its incipient stage. *Figs. 159, 161-163* all show specimens of the disease which is still early. It is important to study these examples to search for a common feature which would account for the phenomenon of pelvic dilatation.

*Fig. 159* is the earliest case of which I have had personal experience. It is the right kidney of a man, age 29, who suffered from attacks of pain in the right side over a period of three years. The attacks at first occurred at three-monthly intervals, but the periods between the attacks gradually shortened until eventually they took place as often as once a week.

The pain suffered by these early cases is often extremely severe. The explanation of this is quite apparent on examining a section of the pelvis under the microscope, for a considerable degree of muscular hypertrophy can be made out. So early a stage of dilatation is

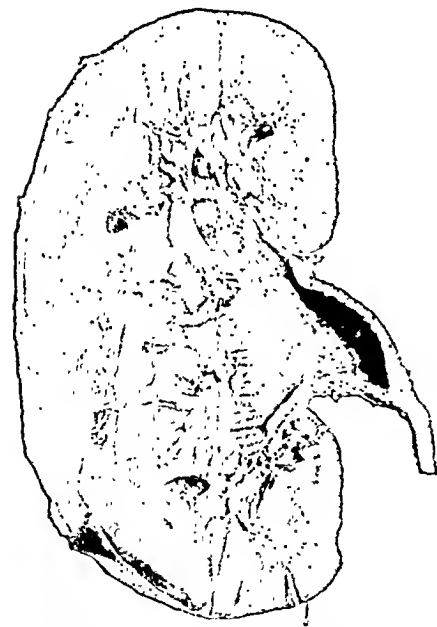


FIG. 159.—An early specimen of hydronephrosis. (Lantern slide.)

represented in the specimen we are considering, that, when handling the organ at operation, it was not at all certain from its appearance that it had been the seat of a general dilatation, so little change was there to be seen in its contour. However, in the knowledge of the pyelographic appearance of this kidney (*Fig. 160*), I had no hesitation in treating it as a hydronephrosis.

*Fig. 161* is also a very early stage of the disease. It is the right kidney of a male, age 28, who had attacks of pain in the right side for eight months. *Fig. 162* is a slightly further stage of dilatation than is shown in either of the

two foregoing specimens. It is the left kidney of a boy, age 15, who had had an operation for acute appendicitis at the age of 13, and who had suffered from intermittent attacks of pain in the left side since.

*Fig. 163* is the left kidney of a woman, age 40, who had suffered from attacks of pain in the left side for three and a half years. Nephropexy was carried out soon after the onset, with temporary relief. Marked thickening of the ureter is obvious in this specimen, and was in fact strikingly present

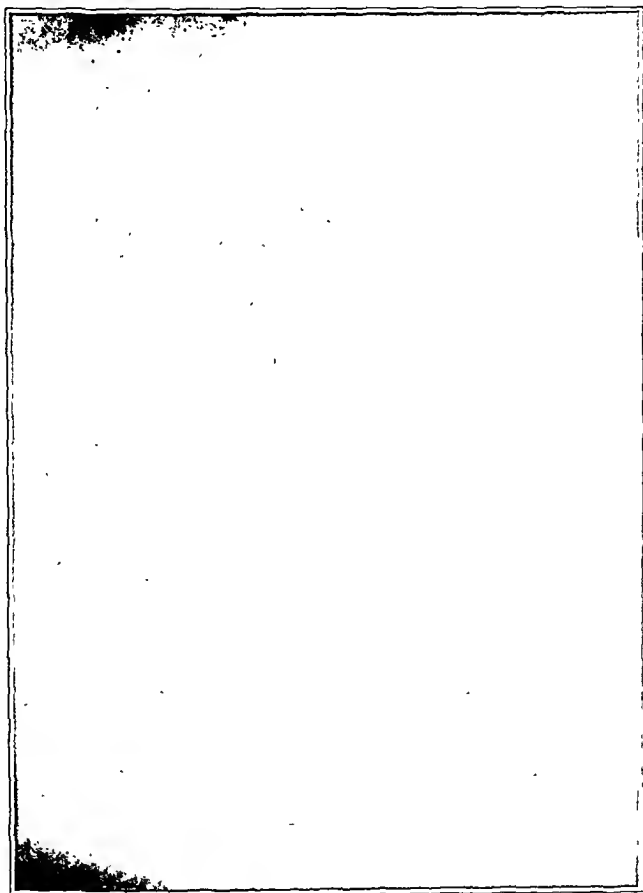


FIG. 160.—Pyelogram of the kidney represented in *Fig. 159*.

in the attached portions of the ureters in all the specimens just discussed. Microscopically this change can be seen to be due to an accumulation of fibrous tissue in the mucous coat (*Fig. 164*). The fibrous tissue has given rise to a definite narrowing of the lumen and is the result of a past infection in the wall of the ureter. The section is from the very early case represented in *Fig. 165*.

The ureters of 14 other specimens showed just the same kind of



FIG. 161.—Another early specimen of hydronephrosis. The dilatation is first noted in the pelves of these early examples.



FIG. 162.—Early hydronephrosis, but slightly more advanced than in the two foregoing examples.



FIG. 163.—Early hydronephrosis, showing very marked thickening of the ureter from inflammation. (Lantern slide.)

change, but in different degrees. One cannot, however, bring forward advanced cases of hydronephrosis, and point to the marked inflammatory changes in the ureter, and maintain that these must be the cause of the hydronephrosis, for it may be said in such instances that the inflammation of the pelvis and ureter is a consequence and not a cause of the dilatation. It is not possible to prove which is the case by investigating the grosser examples of the disease, but the presence of old-standing inflammatory change in the ureters and pelves of the earliest specimens of hydronephrosis strongly suggests that the inflammation is of longer standing than the dilatation. This is the only way to account for a long history of attacks of pain which is sometimes a puzzling feature of cases that are obviously extremely early,



FIG. 164.—Low-power transverse section of upper end of ureter, from specimen shown in Fig. 159. Note the marked fibrotic change in the submucous region, and that this has given rise to constriction of the lumen.

judging from the amount of dilatation. The explanation is that the symptoms at first were due to chronic pyelitis, and later to hydronephrosis or to both of these causes. Take the case represented by *Figs. 159 and 164*. On naked-eye examination it is about as early a stage of dilatation as one is able to recognize, and yet the patient had suffered from his attacks over a period of three years. The microscopic section of the ureter leaves no doubt about the chronicity of the inflammatory change. Geraghty and Frontz, in a detailed examination of the upper end of the ureter in 9 cases of hydronephrosis, found definite narrowing of the lumen in all. In 8 they considered that chronic inflammation was the cause, and in the remaining case hypertrophy of muscle about the pelvic outlet.

In all of the above specimens that I have brought forward it is to be noted that there is a complete absence of any kinking or angulation of the upper end of the ureter. This change, on the other hand, is a common feature of advanced examples of hydronephrosis. It is due to one or both of two causes—namely, an implication of the upper end of the ureter by a renal blood-vessel during the process of pelvic distention, and a sagging of the pelvis below the level of the point at which the ureter joins the peritoneum, for the latter structure gives a certain amount of fixation to the former. The distorted course of the ureter in some of the more



FIG. 165. — Fairly advanced hydronephrosis, showing pelvis and ureter in contact. (Lantern slide.)

advanced cases can often be clearly seen in pyelograms with an opaque ureteric catheter in position. One result of the pelvic sagging is that the pelvis and a certain length of the ureter are brought into intimate contact (*Fig. 165*). This contact is maintained by the formation of adhesions resulting from the spread of inflammation which is present in the ureter and pelvis in all cases. The oblique course thus given to the ureter adds another obstructive process to the pelvic outlet, which becomes oblique and valve-like; further pelvic distention and sagging occur, so that eventually the

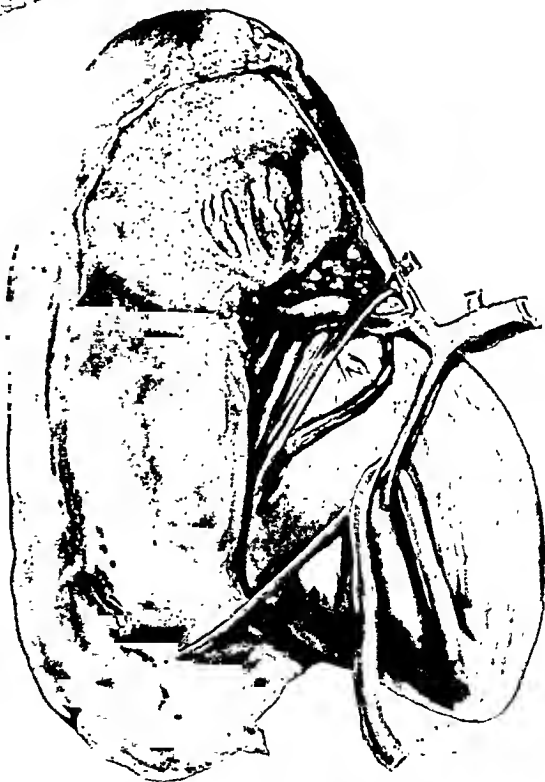


FIG. 166.—Posterior view of a left hydronephrosis. The ureter has become looped over the abnormal venous tributary to the spermatic vein shown in *Fig. 177*.

ureteric orifice, instead of being at the most dependent point of the pelvis, may be found half-way up or higher on the pelvic wall. Many specimens of the most advanced stages of hydronephrosis show the multiplicity of obstructive processes to the urinary outflow from the pelvis that I have just described. Each of these is often quoted singly as the cause of hydronephrosis.

In the most advanced specimen of the disease which I have been able to investigate in detail the following facts were all to be noted: (1) Looping of the ureter over a blood-vessel (*Fig. 166*); (2) Adhesions between pelvis and ureter, with marked obliquity of the upper part of the latter; (3) A contracted valve-like pelvic outlet; (4) Marked inflammatory change in the ureter, with shrinking of the whole of this structure and narrowing of its lumen (*Fig. 170 D*). The extent to which the lumen is narrowed is sometimes best appreciated by inspecting the pelvic outlet, and seeing how stenosed this has become.

It is hardly possible that the inflammatory changes described as being constantly present in the ureters of hydronephroses are not present in the pelvis as well. Of the 18 specimens which I have examined microscopically none failed to show these. There was to be seen in each an accumulation of fibrous tissue, in the mucous region extending often into the muscular coat, and infiltrated with varying numbers of inflammatory cells.

*Fig. 167* is a good example of pyelitis in association with hydronephrosis. The injected areas of the pelvis, situated mostly round the orifices of the minor calices, indicate the regions of most active inflammation. The thickening of the pelvis and ureter—especially at the junction of these two structures—indicates how chronic the condition has been.

It is convenient at this stage to study the series of changes in the ureter when it is the seat of chronic inflammation in association with pyelitis. In the first place there is a general hypertrophy of all the coats, accompanied by a dilatation of the lumen and lengthening of the whole ureter. This is followed by a gradual accumulation of fibrous tissue, commencing in the mucous coat and gradually extending more deeply, so that the ureter becomes



*FIG. 167.* — Hydronephrosis, showing acute pyelitis and thickening of ureter from chronic inflammation.

considerably thickened and still has a dilated lumen. Ultimately, as the mass of fibrous tissue contracts, the whole ureter shrinks and the lumen is seriously constricted.

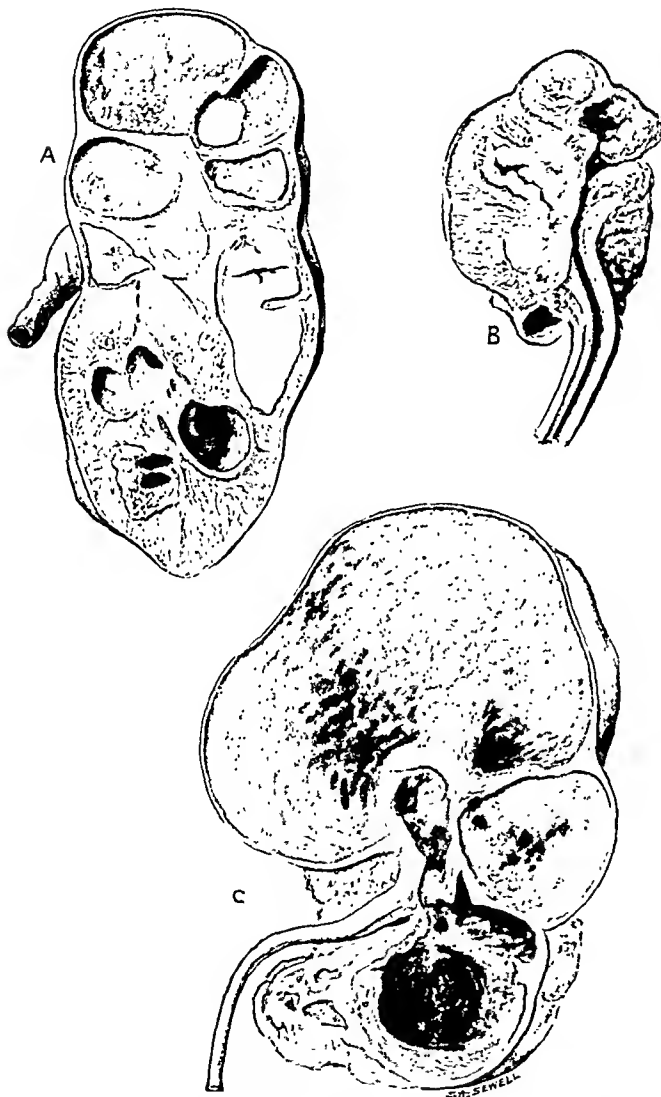


FIG. 168.—Three specimens of tuberculous kidneys, illustrating the three stages of chronic inflammatory change in the ureter. A, Dilatation; B, Thickening; C, Contraction.

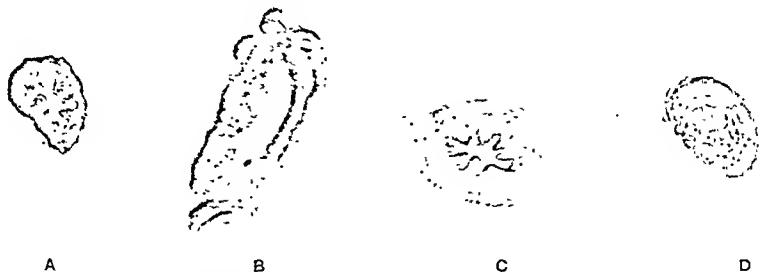
chronic *Bacillus coli* pyelitis are easily and frequently shown by pyelo-ureterogram, the redundancy of the ureter showing itself as a fold at the upper end.

Each of these stages is seen during the progress of renal tuberculosis.\* The first two stages are met with more commonly than the last, because the majority of tuberculous kidneys are removed before the last stage is reached. Fig. 168 illustrates these three stages. In the last example the kidney has been completely destroyed by the tuberculous process, and the ureter reduced to a solid mass of fibrous tissue, so that the lumen could only be made out under the microscope. The dilatation of the ureter which occurs is quite independent of any obstruction to the lower part of the duct. Braasch, Rafin, and Boeckel all call attention to the above-mentioned changes in tuberculous ureteritis. The changes in the ureter corresponding to those of tuberculous disease are equally evident in chronic infection due to causes other than tuberculosis.

The dilatation of the pelvis, and dilatation and elongation of the ureter, associated with early

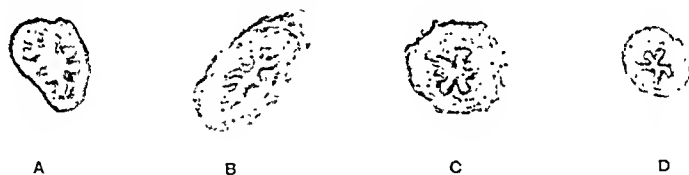
\* I had a recent clinical experience of the dilatation and patency of a tuberculous ureter. After passing a ureteric catheter up to the pelvis of a tuberculous left kidney, the catheter was seen to move regularly up and down the ureter, the oscillations synchronizing with the respiratory movements of the patient.

Dilatation of the ureter is commonly found accompanying the early stage of renal calculus. *Fig. 169* represents cross-sections of various ureters, drawn to scale, illustrating the changes due to chronic inflammation. The first is a normal ureter, the second is from a case of renal calculus. A stone the size of a filbert nut lay in the renal pelvis, and gave rise to a slight degree of



*FIG. 169.*—Transverse sections of ureters showing changes due to chronic inflammation. A, Normal ureter; B, Ureter of chronic pyelitis; C, Ureter from calculous pyonephrosis; D, Ureter from advanced renal tuberculousis.

chronic pyelitis. The whole ureter is hypertrophied and the lumen dilated. The third section is from a calculous pyonephrosis. The kidney substance had been completely destroyed. The wall of the ureter is considerably thickened by infiltration with chronic inflammatory tissue, and the lumen is somewhat narrowed. The fourth is from an advanced case of renal tuberculousis. The kidney had been completely destroyed and no lumen could be made



*FIG. 170.*—Transverse sections of ureters from specimens of hydronephrosis, showing shrinkage of ureter and narrowing of lumen as the disease advances.

out on naked-eye inspection of the ureter. These same changes are to be found in the ureters of pelvic hydronephroses. *Fig. 170* is a series representing these. A is a normal ureter and D is from a more advanced stage of hydronephrosis than either of the two foregoing.

In regarding the whole series, not only does the lumen become progressively smaller, but the ureter shrinks as the disease advances. This reduction in size of the ureter as a whole is a common feature of old-standing cases of hydronephrosis, and results in a general inadequacy of the lumen.

REFERENCES.—19, 23, 24, 25, 32, 50, 53, 61, 111, 118.

### EXPERIMENTAL DEMONSTRATION AND CLINICAL INVESTIGATION OF STRICTURE OF THE URETER.

I have been able to demonstrate experimentally the presence of inflammatory strictures in the upper ends of ureters which macroscopically appeared to be perfectly healthy. The method adopted was to take a freshly



removed post-mortem kidney and ureter of an adult and to inject hot jelly through the divided end of the ureter until the kidney and pelvis reached the limit of their capacity for distention. The whole was then allowed to solidify and fixed in formalin.

In this way 25 kidneys were examined. In 4 there appeared, as the distention reached its limit, a stricture in the region of the pelvo-ureteric junction (*Fig. 171*). In the remainder no such constriction appeared; *Fig. 172* shows some of these. *Fig. 171* reveals clearly four examples of narrowing of the upper end of the ureter. In B and D the narrowing is considerable in both the

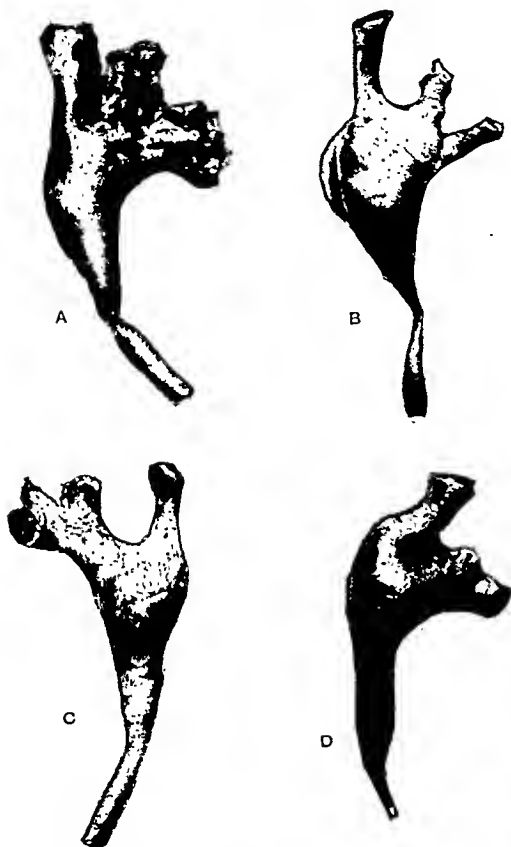


FIG. 171.—Experimental specimens illustrating strictures in the vicinity of the pelvo-ureteral junction.

transverse and long axis of each ureter. A and C show more localized and less marked constrictions. Sections of these specimens through the strictured areas show that the stenosis is due to an accumulation of well-formed fibrous tissue situated in the mucous coat. There was no discernible evidence of any recent active inflammation in any of the regions under investigation, but the appearance indicated in each case the scar tissue of a past inflammatory focus. The presence of these strictures in some cases, and their absence in others, indicates their abnormality.



FIG. 172.—Experimental specimens illustrating the absence of strictures demonstrated in the preceding illustration.

The identification of ureteral strictures at various points along the course of the ureter by means of ureterograms is often quite unreliable, for what appears to be a stricture on one occasion may have completely disappeared at a subsequent examination carried out soon after. Hunner claims to have identified ureteral strictures by means of wax-tipped bougies. Walther has called attention to cases of ureteral stricture associated with calculus in the ureter. Out of 16 cases of such strictures, 9 had previously passed stones.

The site and nature of ureteral stenoses can only be studied accurately by investigations carried out at operations and on specimens of morbid anatomy.

REFERENCES.—34, 67, 123.

### PYELITIS OF PREGNANCY AND OTHER INFLAMMATORY DISEASES AS PREDISPOSING CAUSES.

In view of the important rôle ascribed to inflammation in the etiology of hydronephrosis, a consideration of the evidence indicating any well-marked inflammatory disease occurring previously to the onset of renal symptoms is worthy of some attention. In examining the records of the cases I have collected from the files of the Royal Free Hospital and those I have personally observed elsewhere, I have found 37 cases in which the state of health previous to the commencement of renal symptoms was ascertained. In 32 (86·4 per cent) there was definite evidence of pre-existing chronic inflammation, or some severe acute infection. In 12 cases (35·2 per cent) gastro-intestinal disturbance was the antecedent. In 8 of these the disease was chronic, while in 4 there had been acute appendicitis. In 7 other cases the renal symptoms suggesting pyelitis had their first onset either during or following pregnancy. In 4 cases rheumatic fever had preceded the hydronephrosis. Pneumonia, pulmonary tuberculosis, typhoid, and malaria were noted among the other less common previous illnesses.

The incidence of gastro-intestinal disorders either preceding or accompanying pyelitis raises the question of the route by which the infection reaches the kidney from the intestinal tract. The frequent occurrence both of pyelitis and hydronephrosis as a unilateral disease suggests that the infection takes place by a lymphatic route rather than through the blood-stream. The lymphatic connection between the ileocaecal region and the right kidney is an important one in consideration of the frequent evidence we have of pyelitis complicating acute appendicitis. Hæmaturia as an accompaniment of acute appendicitis is by no means rare, and many cases have been reported. Block holds that the adhesions which are to be found about the pelvis of the kidney following acute appendicitis, by giving rise to obstruction of the ureter, lead to pyelitis, but it is far more likely that the adhesions owe their origin to the spread of inflammation from the pelvis and ureter, the infection of which structures is secondary to the appendicitis. This view is supported by finding the strongest adhesions adjacent to the pelvis and ureter.

It is doubtful whether a peri-ureteritis alone would cause sufficient scar tissue to constrict the ureter. The writer carried out on rabbits two experiments in which 10 per cent iodine was injected retroperitoneally round one

ureter. The animals were killed at nine and twelve months respectively following the experiments. Although considerable scar tissue had formed round each ureter concerned, no constriction leading to hydronephrosis resulted. It has to be borne in mind that while the ureter remains attached to the peritoneum the scar tissue cannot completely surround it, and thus cannot press it from all sides.

REFERENCES.—3, 5, 8, 10, 15, 19, 44, 50, 54, 62, 81, 99, 104, 114, 117, 133.

### PYELITIS, INCREASED FREQUENCY OF MICTURITION, AND POLYURIA.

The association of pyelitis with hydronephrosis has escaped recognition largely because pyelitis is looked upon as a disease which is always associated with pus and bacteria in the urine and with increased frequency of micturition. From my own observations I am of the opinion that the milder degrees of the chronic form of the disease exist with only the scantiest pus and bacterial cells in the urine, and with these absent at certain periods. There is no reason why the inflammation should always go on to the stage of pus formation in this situation more than in any other part of the body. Exacerbations of the existing pyelitis will explain the attacks of pain which come on without apparent cause, and which are a striking feature of nearly all cases of hydronephrosis.

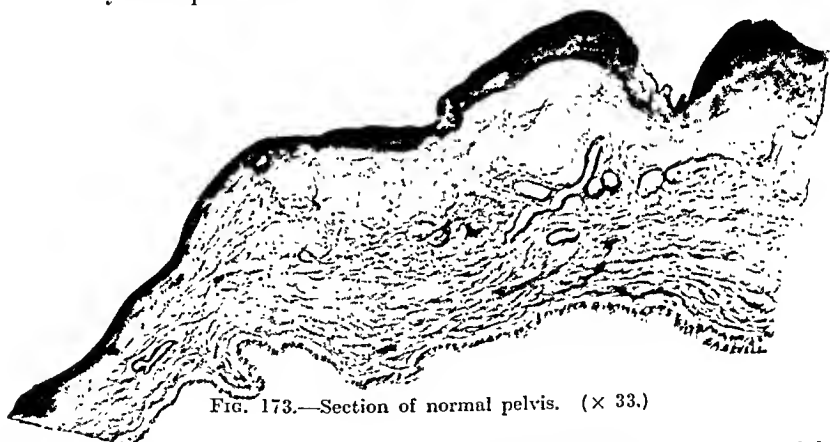


FIG. 173.—Section of normal pelvis. ( $\times 33$ .)

Microscopic examination of the pelvis of eighteen examples of hydronephrosis shows that pyelitis is constant in this disease. *Figs. 173, 174, 175* are sections under the low power of three renal pelves. The first is a normal one, the other two are early cases of hydronephrosis. In the hydronephrosis specimens the muscular tunics are highly developed, and there is a considerable accumulation of fibrous tissue in the mucous and submucous regions. The epithelium in the middle section is in a state of hypertrophy. In the same section the chronic inflammatory tissue has accumulated to such an extent as to give rise to a papilliform process. In the last section the signs of chronic inflammation are not so marked; on the other hand, there is a general infiltration by inflammatory cells, indicating a more acute process than in the former.



FIG. 174.—Section of pelvis of an early hydronephrosis. The chronic inflammatory tissue beneath the mucosa has formed a papilliform process. ( $\times 33$ .)



FIG. 175.—Section of pelvis of an early hydronephrosis, showing a less degree of the same change as seen in the preceding section. ( $\times 33$ .)

It is of some interest to compare the clinical features of these two cases. *Fig. 174* is from a left hydronephrosis in a girl, age 16, who had had pain in the left side for four years. Several examinations of the urine had failed to reveal any bacteria. On one occasion a few pus cells were found. The patient had never suffered from any abnormal frequency of micturition, but on two occasions cystoscopy had revealed slight redness and swelling of the corresponding ureteric orifice. So that in this case with very marked pyelitis the association of inflammation with the hydronephrosis could only be definitely established by cystoscopy. Even this means may fail to reveal any signs of



FIG. 176.—Pyelogram of early hydronephrosis.

inflammation if the examination is not carried out either during or soon after an attack of pain.

The clinical features manifested by the case represented by *Fig. 175* were much more straightforward. The patient, a woman, age 43, suffered from attacks of pain in the right side and frequency of micturition over a period of six months. Several examinations of the urine all revealed considerable pus cells and *B. coli* in the urine. The greater abundance of inflammatory cells in the section of this pelvis is in keeping with the urinary symptoms and findings in this example. *Fig. 176* is a pyelogram of the kidney, and shows

how early and yet how definite the hydronephrosis is. Block calls attention to the association of pyelitis with hydronephrosis, in quoting a number of personal cases.

From a consideration of the two foregoing cases it is apparent that any increase in the frequency of micturition is dependent on the acuteness of the pyelitis. Of 22 cases of hydronephrosis of which I have had personal experience, 50 per cent suffered from increased frequency in some degree, during the periods of the attacks.

**Polyuria.**—It is a common belief that a crisis of hydronephrosis culminates in a discharge of a large quantity of urine from the affected kidney, leading to frequency of micturition from the passage of a considerable measure per urethram within a very short period. This phenomenon, however, is only occasionally to be observed. The number of cases in which polyuria has been definitely established are surprisingly few. One has to bear in mind that there is a danger of misinterpreting the increased frequency and polyuria of pyelitis, which are often present in hydronephrosis. In the records of the numerous cases I have referred to, I can find only 5 instances in which polyuria was satisfactorily established. In the 22 cases of my own personal experience, I could find no evidence that this had occurred.

Polyuria, however, following a renal crisis, is well known to occur in kidneys in which it has been determined by operation that the organ is not the seat of a hydronephrosis. Albarran recorded 3 such cases. In one the attacks had been recurring for four years, and the patient would pass 1½ litres in three micturitions during an hour. At operation the kidney was enlarged from congestion, but there was no dilatation of the pelvis. He has observed hæmaturia in 3 cases of this kind.

That polyuria associated with hydronephrosis is due to just the same cause—namely, the relief of a congestion of a kidney—has been proved by the reports of many observers. These have agreed that the enlargement of the kidney which takes place is due to intense congestion, and that the size of the organ is frequently such that it could not possibly contain the amount of urine voided. Gardner records that a ureteric catheter passed during an attack has revealed not more than an ounce of urine in the pelvis, while Bazy has measured as much as 7 litres of urine in twenty-four hours from a similar case in which there was no pelvic dilatation.

Seeing that pyelitis has been definitely established as an accompaniment of hydronephrosis, there is every reason to believe that the renal congestion which occurs results from the attack of inflammation, which probably reaches its maximum in the wall of the pelvis, but which involves the parenchyma as well.

In reviewing the phenomena that I have described which are known to accompany a renal crisis, especially in a hydronephrotic kidney, it is clear that the congestion and enlargement of the kidney, the presence of redness round the ureteric orifice, and the increased frequency of micturition, all combine to suggest renal infection, even though pus and bacterial cells may be scanty and difficult to detect in the urine. The presence or absence of one or more of the signs or symptoms will depend on the degree of infection. In view of the constant evidence of nephritis, pyelitis, and ureteritis as revealed

by the microscope in these cases, it is impossible not to consider most seriously infection as the cause which precipitates an attack.

REFERENCES.—2, 3, 5, 17, 19, 21, 22, 23, 33, 44, 50, 52, 53, 54, 60, 63, 66, 75, 77, 79, 91, 128, 132.

### RENAL MOBILITY.

Following the writings of Landau, of Terrier and Baudouin, and of Tuffier, renal mobility as the cause of hydronephrosis was for many years accepted without question. The events which were supposed to form the cycle leading up to a crisis seemed so obvious as to render criticism superfluous. Gradually, however, successive facts have been brought forward which have combined to show up fallacies in the old arguments, with the result that the theory of renal mobility as a cause of hydronephrosis has been almost completely discarded. I can find no record of any painstaking observations carried out by a modern investigator which does not discredit the theory of mobile kidney.

Briefly the events of the cycle credited to this theory are as follows: Owing to the mobility of the kidney the upper part of the ureter becomes kinked; this causes a retention of urine in the renal pelvis, with resulting pain and enlargement of the organ; subsequently the obstruction is overcome, when the discharge of a large amount of urine occurs, with relief of pain and diminution in size of the kidney. At first sight this explanation seems extremely reasonable, and one does not wonder that it was so readily accepted. However, the consensus of opinion now is that kinking of the ureter, to produce obstruction, must be long-continued, and that to bring this about fixation of the upper end of the ureter is an essential. But as the ureter is a flexible and elastic tube, kinking does not diminish the size of the lumen. Experimentally it has been shown that an acute bend in the ureter as a result of suturing together its walls does not produce a change in the renal pelvis. In post-mortem observations on the kidney I have been impressed with what appears to be the impossibility of producing an acute bend in the ureter, even after freely mobilizing the kidney as it lies *in situ*.

There is, however, no doubt that renal mobility is commonly found in association with hydronephrosis. The abnormal range of movement is, of course, a consequence of the hydronephrosis and not the cause of it. The variations in size of the organ give rise to a stretching of the fibrous sheath of the fatty capsule and of the vascular pedicle which runs across the front of the dilated pelvis.

Tuffier, in his experiments on dogs in which he attempted to cause hydronephrosis by mobilizing the kidney, could only kink the ureter by passing a thread round it. Legueu, Hildebrand, and Haga all demonstrated that the upper ureter must be fixed in order to produce obstruction by kinking this structure. Numerous cases of mobile kidney have been operated upon in which neither kinking of the ureter nor dilatation of the pelvis was to be made out, and many others have been investigated by ureteric catheterization and pyelography with the same result.

*Fig. 157* makes an interesting study. There is a hydronephrosis involving the lower pelvis and its calices of a kidney with a double ureter. The kinking

of the commencement of the lower ureter is quite obvious. If it is to be assumed that this is due to a dropping of the whole organ from mobility, the upper ureter should be equally affected. The most acceptable explanation is that the redundancy of the ureter which has occurred in the early stage of inflammation, and the dilatation of the pelvis which has taken place later, have allowed these two structures to come into contact.

Henry Morris, in giving an account of 142 cases of hydronephrosis, is reported as saying: "I am much disposed to think that in several cases in which hydronephrosis and movable kidney coexist, the mobility was acquired after and not before the hydronephrosis".

The Dietl's crises sometimes to be observed in cases of movable kidney are no doubt due to renal retention, not from an obstructed outlet of the pelvis, but from a congested kidney resulting from a pyelitis consequent on chronic intestinal trouble so often present with general visceroptosis, of which the nephroptosis is a part. Many cases having long histories of crises fail to show any indication of a dilated pelvis when submitted to operation. Tuffier, in describing the results of 45 nephropexies for attacks of pain, could find no evidence of dilatation in 36. Geraghty and Frontz quote the combined figures of seven authorities, giving a total of 4576 cases. These show that mobile kidney occurs in 20 per cent of women and in 2 per cent of men. They also refer to Kelly and Burnham on 245 cases. They found the mobile organ on the right side in 177 cases, on the left in 25, and on both in 43. The writers go on to point out that if mobile kidney were the cause of hydronephrosis, we should find an overwhelming majority in women, right hydronephrosis more common than left, and double more common than left. None of these facts, however, is found to be the case. For the disease is but twice as common in women as in men, it occurs with equal frequency in either kidney, and the bilateral disease is rare. Moreover, mobile kidney is a condition with a marked preponderance of its incidence in the fourth decade, while it is during the third decade of life that most cases of hydronephrosis occur.

REFERENCES.—17, 39, 43, 45, 50, 53, 77, 79, 81, 120, 124, 127.

### TRAUMATISM AND HYDRONEPHROSIS.

The relationship of traumatism to the kidney or ureter in connection with hydronephrosis has been given positions of widely varying importance according to the views of different writers, but in reading the accounts of the majority of cases reported as traumatic hydronephroses it is seen that the diagnosis is reached merely on the detection of a cystic swelling in one kidney region, in a number of cases not many days after the receipt of injury. In some, repeated aspirations have sufficed to cure the condition.

In order to understand the position clearly it is essential to have an accurate knowledge of the pathological anatomy commonly seen in injured kidneys. Transverse fissures in varying numbers and extent, running from the hilum to the outer border, with the maximum width of the fissure at the hilum, is the commonest characteristic. It is frequently found that a fissure has involved the pelvis as well. Guterbock reported a torn pelvis in 40 per cent of 32 examples of injured kidneys found at autopsy. There are probably



many instances of one or more of the larger calices being opened in others. Thus urine will be extravasated into the perirenal capsule in such cases, and soon this sac will become a large cystic swelling. It is said of some of the earliest cases that the ureter becomes blocked with clot, and thus the kidney becomes distended. A knowledge of the results of experimental obstruction to the ureter shows clearly that the capacity of the kidney to become dilated

into an enormous cyst within a few weeks or a much shorter time does not exist. From studies made on specimens of intermittent hydronephrosis, and with a knowledge of the clinical history of such cases, it is clear that only after obstruction has existed for a considerable lapse of time does the renal sac become distended to hold more than a pint of fluid. Nevertheless one frequently comes across examples in the literature of such cases as the following, which is put forward as a traumatic hydronephrosis: Two and a half months after injury to the left side, 6 quarts of fluid were evacuated by incision, from the left renal region of a man, age 44. No description of the pathological anatomy of the region was given. Similar cases are numerous.

Cabot says that so close

is the similarity in size, position, consistency, and mobility that no case of traumatic hydronephrosis can be accepted as an instance of true hydronephrosis unless proved by dissection at operation or autopsy. He describes a case of pseudo-hydronephrosis with a ruptured and collapsed kidney lying at the bottom of the sac.

It is certain that for a true traumatic hydronephrosis to occur, an injury to the ureter is necessary. Dilatation of the kidney will take place only after the formation and contraction of scar tissue in the wall of the ureter. Should the injury be so severe as to cause a complete rupture of the ureter, there will result a peri-ureteral urinary cyst.

Amongst numerous cases reported as traumatic hydronephroses which I have looked up, in two only was the pathological anatomy properly investigated and the lesion of the ureter identified. One was that of a woman, age

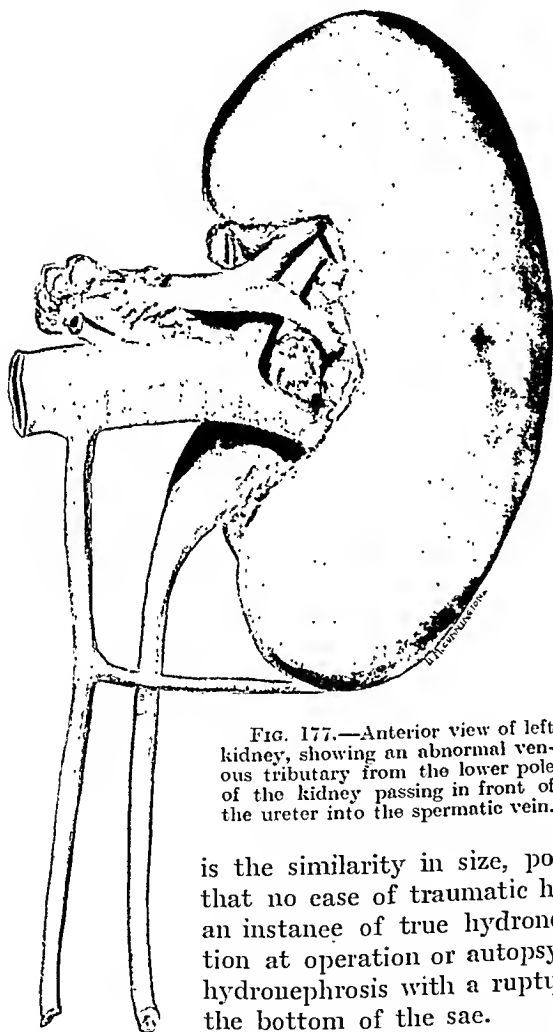


FIG. 177.—Anterior view of left kidney, showing an abnormal venous tributary from the lower pole of the kidney passing in front of the ureter into the spermatic vein.

26, in whom the lumen of the ureter concerned was completely obliterated by scar tissue two months after receipt of injury. The other was that of a man who was struck in one lumbar region by a piece of shell, and in whom ten years later a stricture of the ureter was identified as the cause of a hydronephrosis, which was then palpable.

One writer assumes that trauma is a common factor in the causation of intermittent hydronephrosis, and depends upon the fact that injury to the nephrocolic ligament allows nephroptosis to occur. This view, however, is not supported by any facts of pathological anatomy, but the writer quotes 7 cases out of 18 hydronephroses with a previous history of trauma.

REFERENCES.—S, 49, 50, 63, 71, 75, 80, 125, 127.

### NORMAL AND ABNORMAL RENAL VESSELS IN RELATION TO HYDRONEPHROSIS.

A vascular complication of a hydronephrosis wherein the ureter is obstructed by a blood-vessel is of not infrequent occurrence. There are two sharply opposite points of view with regard to the rôle which these blood-vessels play. I have studied this complication in 11 examples illustrating the condition. These examples

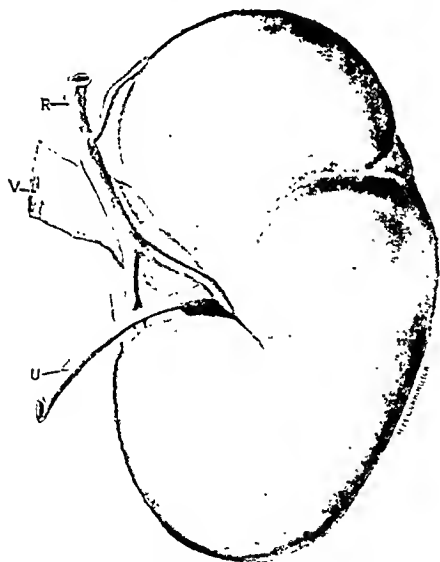


FIG. 178.—Posterior view of a right kidney, showing the normal inferior branch of the renal artery crossing in front of the upper end of the ureter. R, Renal artery; V, Vein; U, Ureter.

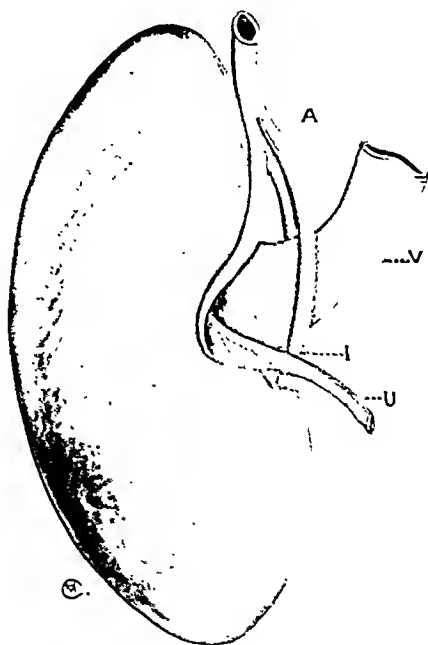
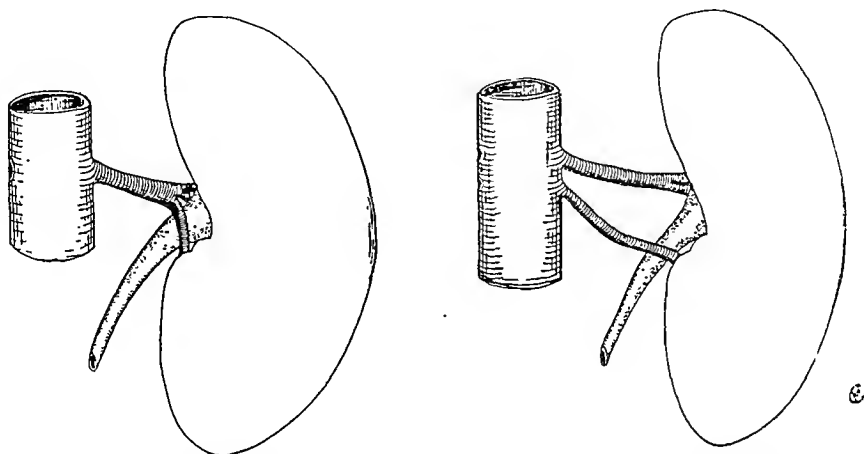


FIG. 179.—Posterior view of a left kidney, showing the same relationship between the normal inferior branch of the renal artery and the upper end of the ureter as in the preceding illustration. A, Anterior branch of artery; V, Vein; I, Inferior branch of artery; U, Ureter.

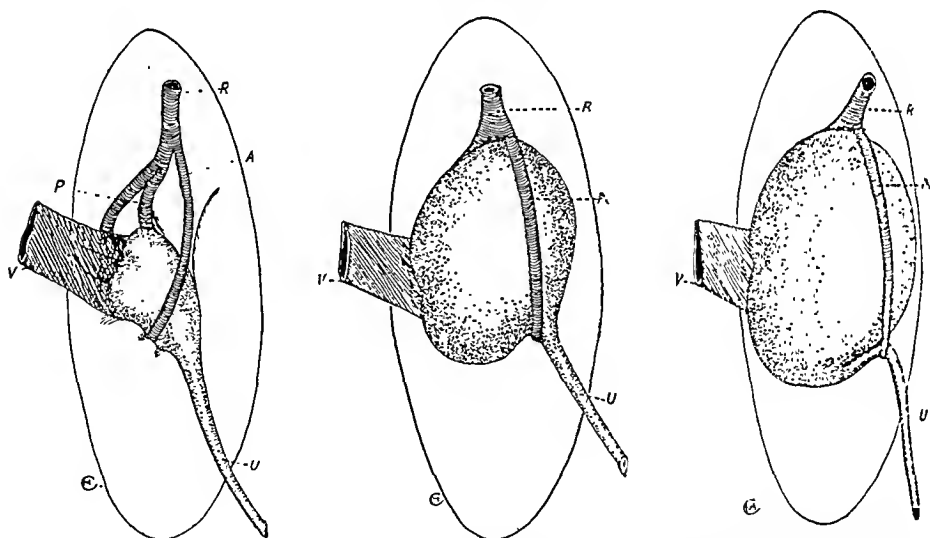
represent all the available local specimens in so far as my searches carry me through the various pathological museums of London. Before discussing

each of these individually, I will consider the normal arrangement of the blood-vessels in the hilum of the kidney.

I have dissected these vessels in 35 specimens and find that the common arrangement is for the renal artery to proceed towards the upper part of the hilum and there to divide into three main branches, two superior, which enter



FIGS. 180, 181.—Showing the extreme variations in origin of the inferior branch of the renal artery.



FIGS. 182, 183, 184.—Medial views of a series of renal hila. The renal vein has been turned forward to show the changing relationships between the pelvis, inferior artery, and ureter during the process of pelvic distension. R, Renal artery; A, Antero-inferior branch; P, Postero-superior branch; V, Vein; U, Ureter.

further into the hilum above the pelvis, and one inferior, which enters the lower part of the hilum by coursing downwards and outwards across the front of the pelvis. It will be seen that it is this normal inferior vessel, either venous or arterial, which commonly plays the important part of compressing

the ureter during the process of pelvic distention. From the relationship which both the inferior artery and vein have to the pelvis as seen in *Fig. 177*, a hydronephrosis would develop and progress without the ureter being implicated in any way by either of these vessels. In *Fig. 178*, which is a posterior view of a right kidney, the inferior artery enters the hilum by passing in front of the upper part of the ureter, thus allowing the pelvis to lie opposite the vascular space bounded above by the superior vessels and below by the inferior arterial branch. *Fig. 179* is a posterior view of a left kidney where a similar relationship exists.

When the pelvis dilates it expands in all directions, but forwards more than backwards in conformity with the direction of least resistance. Should the pelvis in such circumstances lie opposite to the vascular interval just described, it will project into this gap, and in so doing the upper end of the ureter may be drawn with it and so compressed between the inferior artery and the distended pelvis, provided the vascular space is sufficiently large. The size of the space is obviously smallest when the inferior vessel arises from the parent trunk close to the kidney, and becomes progressively larger as this vessel takes its origin nearer to the origin of the parent stem. The interval is largest when the inferior artery arises actually from the aorta itself, as it not uncommonly does. These points are illustrated in the diagrams, *Figs. 180 and 181*.

*Figs. 182, 183, 184* are a series of medial views of renal hila, and represent the changing relationship of pelvis, ureter, and inferior artery during the development of a hydronephrosis. The renal vein is turned forward, and in the first of the series a slightly dilated pelvis is seen, with the two superior arteries above, and the inferior branch passing in front of the pelvis. In the next one the distention has gone a little further, and the ureter has approached nearer to the inferior artery. In the last of the series the ureter is seriously compressed between the artery and the distended pelvis. The artery, which originally lay in front of the pelvis, is now behind that structure. Such a relationship explains the belief that the ureter becomes compressed by an aberrant vessel which passes behind the renal pelvis.

The actual specimens which I have collected will now be considered. Five are from the Museum of Guy's Hospital, and one has been taken from the Museums of each of the following Hospitals: St. George's, St. Mary's, St. Thomas's, the Royal Free, and one from the Museum of the Royal College of Surgeons. I wish to take the opportunity

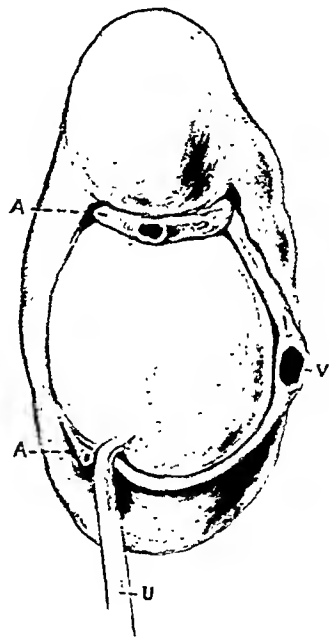


FIG. 185.—Medial view of a left hydronephrosis in which the commencement of the ureter has been kinked by the normal inferior branch of the renal artery. A, Artery; V, Vein; U, Ureter. (From Guy's Hospital Museum, No. 1693.)

of thanking the curators of the Museums concerned for allowing me facilities to have these specimens illustrated.

*Specimen 1.*—Fig. 185 is a medial view of a left hydronephrosis. On the front of the distended pelvis, which is bulging slightly more forwards than backwards, lies the renal vein, of which the inferior tributary can be clearly seen. This vessel is normal in its point of exit from the renal hilum and its junction with the parent vessel. The two superior branches of the renal artery are seen entering the hilum above the pelvis, and their point of origin from the renal artery is evident.



A slight but definite kinking of the upper end of the ureter can be seen, and from the position of the remains of the inferior artery and from the presence of the vertical groove on the inner aspect of the pelvis, it is clear that this kinking was caused by contact with the normal inferior branch of the renal artery.

*Specimen 2.*—Fig. 186 is a postero-medial view of a right hydronephrosis. One sees the forward bulging of the distended pelvis and the inferior vessels passing into the lower part

FIG. 186.—Postero-medial view of a right hydronephrosis, in which the normal inferior branch of the renal vein is compressing the commencement of the ureter. A, Artery; V, Vein; U, Ureter. (From Guy's Hospital Museum, 1920/63.)

of the hilum. There is obviously some commencing interference with the upper end of the ureter by the inferior vein, which is normal in its connection with the main venous renal trunk and in its point of exit from the kidney. The inferior artery, as in the last specimen, has been divided, but sufficient of it remains to indicate that it was a normal vessel. It is the vein in this case which appears to be in most intimate relationship with the ureter.

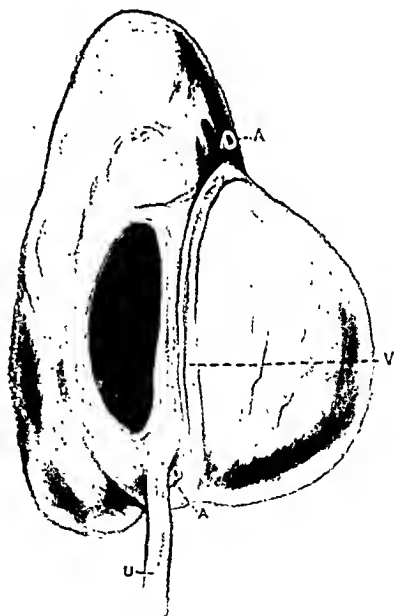


FIG. 187.—Posterior view of a left hydronephrosis. (From Guy's Hospital Museum.)

*Specimen 3.*—Fig. 187 is a postero-medial view of a left hydronephrosis. The inferior vessels are both seen. Neither is compressing the ureter, although each is in the vicinity. The inferior artery has been divided, but the course of its companion vein suggests the normal origin of the artery. It should be noted what a markedly posterior position on the pelvis the inferior vein occupies. In explanation of this,

I would call attention to the considerable forward projection of the pelvis, for the more this structure advances, the more posterior are the inferior vessels likely to become.

In this specimen these vessels are so markedly displaced that one wonders whether the present relationship was not already in existence before the dilatation occurred. If such were the case, it would be necessary that the vessel after passing downward behind the pelvis should curve forward round the inner border of the ureter, and enter the hilum after passing in front of this structure, as indicated in *Fig. 188*. Such a vascular anomaly, as far as I know, has never been described, and probably has never existed, but it is the only possible alternative to the explanation I have given.

It will be noticed that in each of the foregoing examples the inferior artery has been divided by the surgeon close to its entry into the hilum. It would be obvious to the operator in each case that the vessel was lying somewhat posterior to the pelvis, but only by taking additional pains—often considerable on account of the pelvic distention, and unnecessary from the point of view of the nephrectomy—could

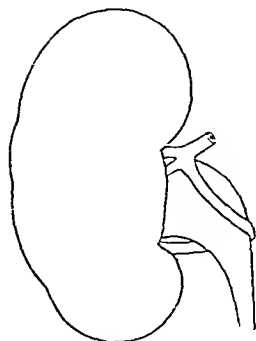


FIG. 188.—Posterior view of a left kidney, illustrating the kind of arterial anomaly that would be necessary were the relationship seen in the foregoing specimen pre-existent to the dilatation.

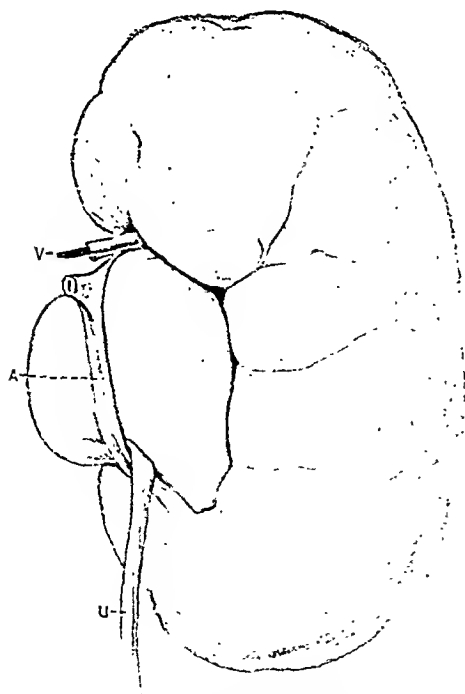


FIG. 189.—Posterior view of a right hydronephrosis, showing compression of the ureter by the normal inferior branch of the renal artery. V, Vein; A, Artery; U, Ureter. (From Guy's Hospital Museum, No. 1695.)

the true origin of the vessel be established. Hence the false theory of hydronephrosis due to an abnormal vessel passing behind the pelvis and obstructing the ureter has arisen, and continues to receive support, for the condition I have described is a common one, and fresh examples are constantly being reported and misinterpreted.

*Specimen 4.*—*Fig. 189* is a posterior view of a right hydronephrosis. The artery which is the offending member is seen to be perfectly normal in origin and point of entry into the kidney. One can imagine in this example that by pulling the ureter downwards, so that the pelvis and upper part of the ureter were drawn backwards, the inferior artery and the pelvis would be restored to their normal relationship—namely, with the artery in front of the pelvis.

*Specimen 5.*—*Fig. 190* is a posterior view of a left hydronephrosis. As in the previous example, the ureter is implicated by the normal inferior vessels, and here, as before, by pulling on the ureter the original relationship of the inferior vessels to the front of the pelvis could be restored.

*Specimen 6.*—Fig. 191 is a posterior view of a left hydronephrosis. The amount of pelvic distention here is very considerable, with a marked forward projection over the normal inferior artery, which is seen lying on the middle of the posterior aspect of the pelvis. The ureter has been dragged forward and has become markedly compressed.

*Specimen 7.*—Fig. 192 is a postero-medial view of a left hydronephrosis. Here also the pelvis has gone well forward, and the ureter has become involved, in this case by the normal inferior branch of the vein.

*Specimen 8.*—Fig. 193 is an antero-medial view of a left hydronephrosis. Here the inferior vein can be seen interfering with the upper end of the ureter. The corresponding artery has an independent origin from the aorta, and those who attach so much importance to such an origin must realize that the normal vein is playing a more important part than the abnormal artery.

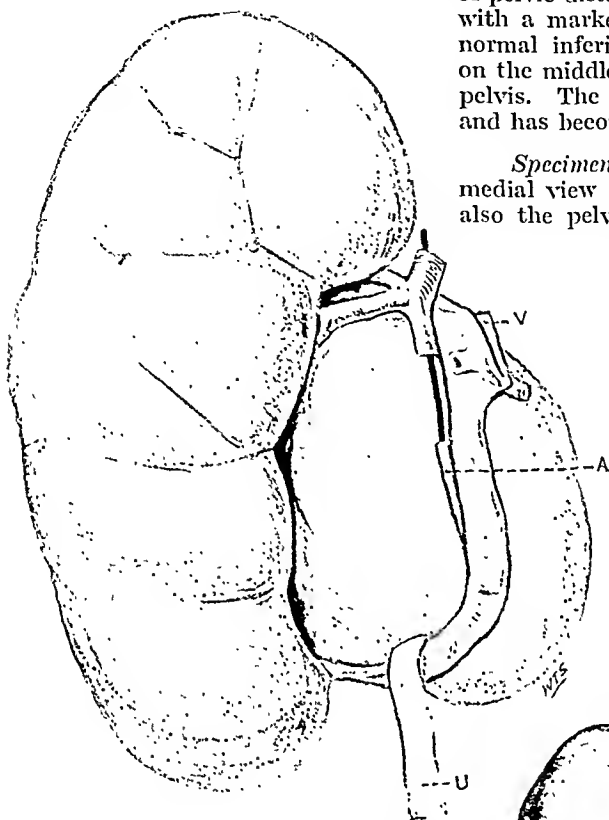


FIG. 190.—Posterior view of a left hydronephrosis, showing a looping of the ureter over the normal inferior branch of the renal vein. V, Vein; A, Artery; U, Ureter. (From Guy's Hospital Museum, No. 1694.)

The interval between the upper and lower branches of the vein is not sufficient to allow the pelvis to bulge through to any great extent. Were the limiting influence of this venous circle not present, no doubt the aberrant artery would have produced a considerable looping of the ureter.

*Specimen 9.*—Fig. 194 is a posterior view of a right hydronephrosis. The inferior artery has its origin close to the aorta and has thus provided a very wide interval between the upper and lower arterial branches. As a consequence the pelvis has gone completely forward and a marked looping of the ureter has

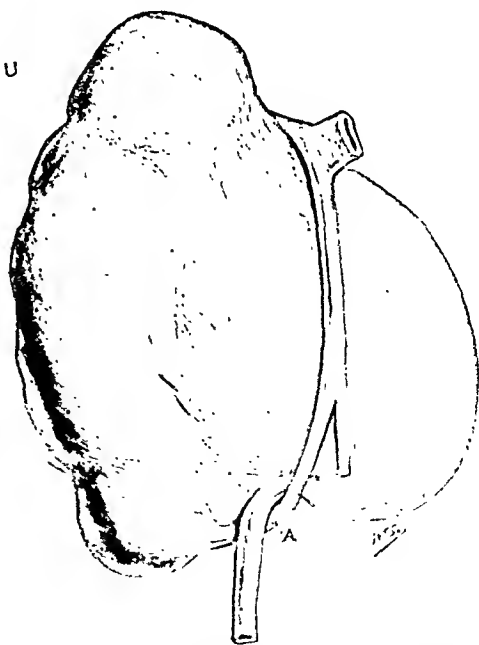


FIG. 191.—Posterior view of a left hydronephrosis. The pelvis has bulged forward over the normal inferior artery, which is compressed the ureter. (From St. George's Hospital Museum.)

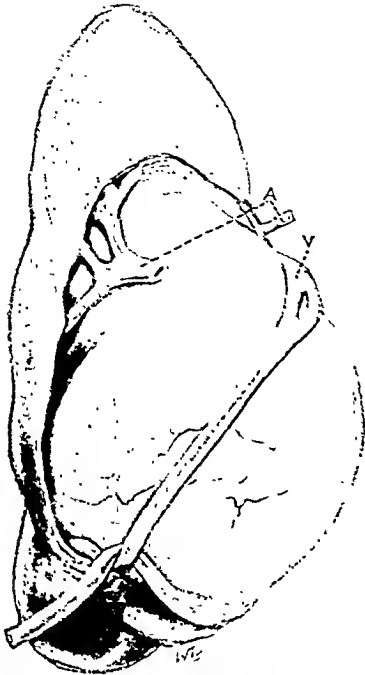


FIG. 192.—Postero-medial view of a left hydronephrosis. The pelvis has bulged forward through the interval between the superior and inferior branches of the renal vein. The inferior branch, which is quite normal, is compressing the ureter. A, Artery; V, Vein. (From St. Mary's Hospital Museum.)

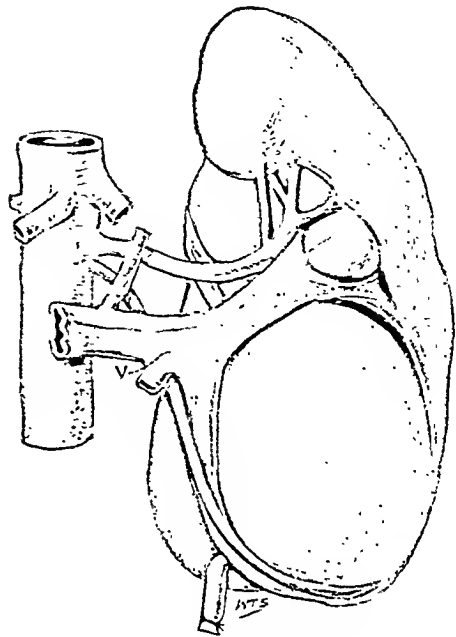


FIG. 193.—Antero-medial view of a left hydronephrosis. The ureter is compressed by the normal inferior branch of the renal vein. (From St. Thomas's Hospital Museum.)

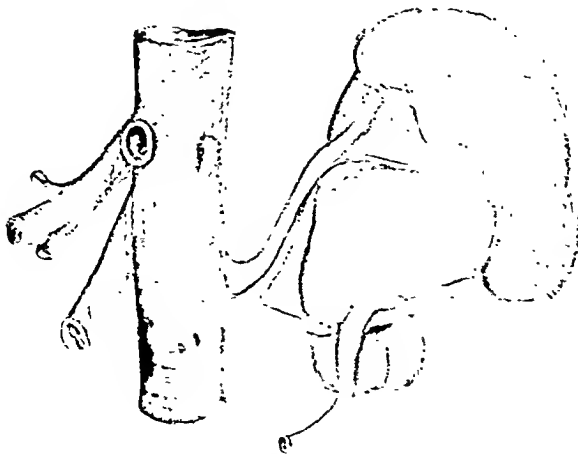


FIG. 194.—Posterior view of a right hydronephrosis. The ureter is looped over the normal inferior branch of the renal artery. (From the Royal Free Hospital Museum.)



taken place. That portion of the ureter which is proximal to the point of crossing of the artery is dilated, indicating the secondary obstruction which has arisen at this point. A slighter dilatation is noted for a short distance just beyond this, however, and no doubt it was here that the original obstruction arose. I have had an opportunity of examining this portion of the ureter under the microscope, and find it to be the seat of chronic inflammation which has resulted in narrowing of the lumen.

*Specimen 10.*—*Fig. 195* is a posterior view of a right hydronephrosis. The pelvis has worked forwards between the superior artery and the inferior. The ureter is being compressed by the latter vessel, which has an independent origin from the aorta. This specimen represents the only genuine example amongst those I have put forward in which the ureter is being compressed by an abnormal artery.

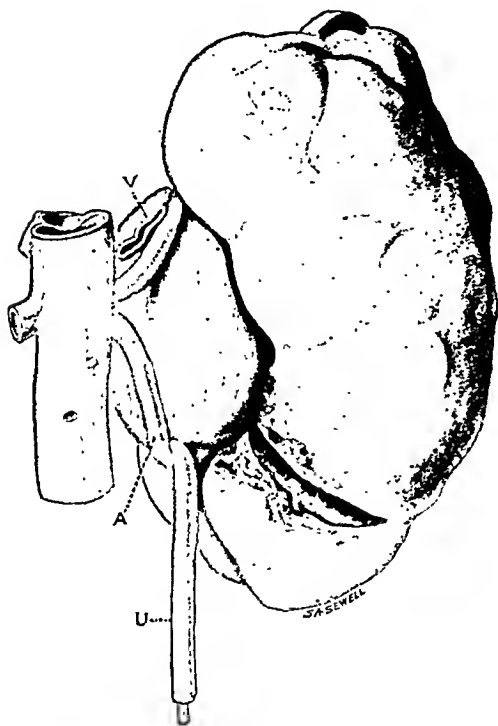


FIG. 195.—Posterior view of a right hydronephrosis. The ureter is compressed by the inferior branch of the renal artery, which is abnormal in the sense that it has an independent origin from the aorta. V, Vein; A, Artery; U, Ureter. (From the R.C.S. Museum, No. 3521b.)

*Fig. 196* is an anterior view of a left hydronephrosis, illustrating an example in which the interval between the superior and inferior groups of renal vessels is not sufficiently large to allow the pelvis to come forward by this route.

I should like to call attention to yet another form of vascular involvement of the ureter. In certain cases there is a venous tributary to the left spermatic vein from the lower pole of the left kidney. The vein crosses superficially to the ureter, as seen in *Fig. 177*. Should the left kidney become the seat of a hydronephrosis in such a case, a marked kinking of the ureter over this venous tributary is likely to result.

*Specimen 11* (see *Fig. 166*).—This is the posterior view of a left hydronephrosis where the complication mentioned above has actually taken place. The vein involving the ureter is seen to join the spermatic vein. The length of ureter proximal to the actual looping is much more than in any of the other examples. This is

explained by the lack of obstruction to the forward progress of the pelvis resulting from the exceptionally wide vascular interval between the aberrant vein and the vessels above.

There is no doubt that some interference with the ureter by a renal vessel is exceedingly common in hydronephrosis. This is borne out by the fact that Mayo, in reporting on 27 cases, found some compression of the ureter by a blood-vessel in 20, and in all but 2 the offending arteries came from the renal trunk.

Among the numerous references in the literature to hydronephrosis there are very many in which an aberrant vessel is cited as the cause. In the large majority there is no detailed account of the actual origins of the vessels or of the points at which they leave the kidneys. In noting this lack of detail one bears in mind how difficult it is during an operation on a distended kidney to be sure of the origin of certain vessels. Papin and Iglesias call attention to this point. No doubt this difficulty has often led to the erroneous conclusion that a normal vessel is an abnormal one. Kuster, Israel, Bazy, Duval, Grégoire, and other able observers all reject the abnormal-vessel theory.

In a number of reported cases there is an illustration of the diseased kidney. In each one that I have seen there exists the same relationship between pelvis, ureter, and blood-vessels as was apparent in the series I have brought forward; moreover, although in many instances the vessel is described as abnormal, it is obviously a normal one.

Bazy gives a diagrammatic illustration of a different view of the development of the vascular complication. He shows the inferior vessel passing behind the ureter. He explains that the dilating pelvis sinks behind the artery, dragging the ureter with it. It must be a rare form of the disease, as none of my specimens shows it. It is difficult to understand in what circumstances the enlarging pelvis should not follow the line of least resistance, which is forwards and not backwards.

Marion supports the view that abnormal vessels are a cause of hydronephrosis, because in his cases he has found the vessel at the level of the constriction of the ureter. The answer to this is that the same relationship is found where the vessel concerned is a normal one. The same view has also a certain amount of support from the relief gained in certain cases after division of the constricting vessel. In this connection, however, it is instructive to turn to the report from the Mayo Clinic dealing with 503 cases of hydronephrosis operated upon between 1907 and 1923:—

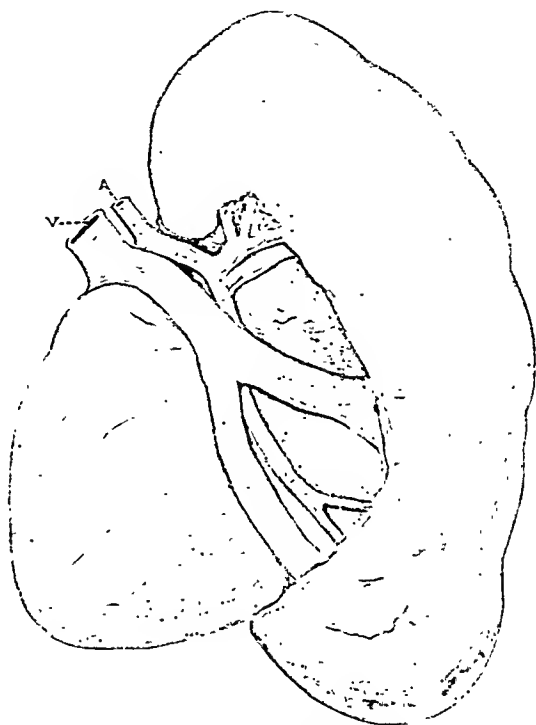


FIG. 196.—Anterior view of a left hydronephrosis, showing the relationship when the interval between the superior and the inferior branches of the renal vessels is not large enough for the pelvis to project through.

In 17 cases cutting of anomalous blood-vessels was carried out, in 10 cases with a satisfactory result, and in 7 with an unsatisfactory result. In 9 cases nephropexy was carried out as well, with unsatisfactory results in 3 cases. The patients who were relieved by division of the blood-vessels were those whose symptoms were only moderate, or who had only a slight degree of dilatation.

In view of the definite narrowing which has occurred at the pelvic outlet in many of the more advanced cases, it is difficult to see how any cure could result in any but the earliest examples. On the other hand, in those cases in which there is a definite compression of the ureter on to the dilated pelvis there must obviously be some relief to the obstruction by dividing the vessel.

REFERENCES.—4, 6, 15, 16, 17, 20, 30, 31, 33, 38, 41, 50, 53, 58, 65, 72, 78, 79, 80, 82, 86, 87, 88, 97, 103, 105, 108, 121, 122, 128, 135.

### SUMMARY.

1. Hydronephrosis with the dilatation beginning at the pelvo-ureteral junction is the commonest form of the disease. It occurs both as a congenital and an acquired condition.

2. Many cases of hydronephrosis have been reported in abnormally developed kidneys, but in what way these predispose to the disease is not known.

3. In a large proportion of the cases identified in foetal and early child life, and which are undoubtedly congenital, the dilatation is bilateral and often involves one ureter or both ureters, while in some foetal examples the lumen is completely obliterated.

4. In the great majority of cases of hydronephrosis the first manifestations of the disease are in adult life, most commonly between the ages of 20 and 30 years. In many of these the kidney is found to be in an extremely early stage of dilatation. It is therefore assumed that such cases represent the acquired form of the disease.

5. The earliest cases show narrowing of the ureter from chronic inflammation, and this appears to be the cause of the dilatation. In all cases there is an associated pyelitis, and some chronic interstitial nephritis.

6. Exacerbations of the existing pyelitis will explain the attacks of pain which come on without apparent cause in most cases of hydronephrosis. There is evidence of the infection either on cystoscopy or in the urine in a large proportion of cases during an attack of pain, which is often accompanied by increased frequency of micturition. There may be little or no evidence of infection after an attack has passed.

7. Chronic, or severe acute, inflammatory disease in any part of the body, chronic gastro-intestinal derangement, and the pyelitis of pregnancy are common antecedents of hydronephrosis.

8. There is no conclusive evidence that renal mobility is ever a cause of hydronephrosis.

9. Traumatism is a rare cause, and results from the contraction of scar tissue in the wall of an injured ureter.

10. In drawing conclusions from the specimens I have put forward illustrating a vascular complication of hydronephrosis, I would call attention to the following facts :—

a. They represent all the available examples demonstrating a vascular complication of a hydronephrosis, and throughout the whole series there is a complete uniformity in the mechanism producing this complication.

b. In 9 of the 11 examples the vessel implicating the ureter is a normal inferior branch of one of the main renal vessels. In some instances it is the venous and not the arterial branch. In none of these specimens is the artery implicating the ureter aberrant in the sense that it enters the kidney through the cortex and not by way of the hilum.

c. It is therefore concluded that the process here described is the usual one which determines a vascular complication of the ureter in certain cases of hydronephrosis. The aberrancy of the vessel concerned is not necessary, in fact occurs infrequently, but offers the most favourable opportunity for compression of the ureter to occur when present.

d. It is obvious, however, that ultimately the ureter may be seriously compressed between the vessel and the distended pelvis in certain cases, and therefore some relief is likely to result from division of the constricting vessel.

In conclusion, I wish to express my thanks to the Surgical Staff of the Royal Free Hospital, to each member of which I am indebted for material for this paper, and also to Dr. H. M. Cunningham for her assistance in the experimental work on rabbits, and in some of the histological work. Help from Mr. Ulysses Williams and Dr. Scott Williamson is also gratefully acknowledged. I have to divide my thanks between Dr. Cunningham, Mr. Thornton Shiells, and Mr. S. A. Sewell for the excellent illustrations that they have provided.

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## TUMOURS OF THE TESTICLE : THE SPERMATOCYTOMA GROUP.

BY F. GORDON BELL, EDINBURGH AND DUNEDIN.

IN a previous paper published in THE BRITISH JOURNAL OF SURGERY for July, 1925, the teratoid group of testicular new growths has been described. Before passing on to consider the second relatively common type of neoplasm—the seminiferous or germinal-celled tumour, called variously spermatocytoma, seminoma, round-celled sarcoma, and embryonal carcinoma—it is necessary to give a brief account of the development and normal structure of the testis, as indicating lines of pathogenesis, more particularly of the seminiferous tumours.

**The Secretory Apparatus of the Testis.**—The early phases in the development of the human testis have still to be satisfactorily elucidated. We are concerned here more particularly with the origin of the seminiferous cells, about which two conceptions prevail:—

1. It is generally stated that the seminiferous cells are derived from the columnar germinal epithelium (mesothelium) which covers the genital ridge lying medial to the Wolffian body. This specialized epithelium is believed to grow down in the form of rod-like ingrowths into the subjacent mass of mesodermal tissue (mesenchyme) which outlines the future body of the testis. The surface connections are then lost and the germinal cells appear as solid anastomosing cords transversing the mesoderm. Some of the germinal epithelial cells are characterized by their large size and different staining reactions, and constitute the spermatogonia or primitive sex cells.

2. There are, on the other hand, the elaborate researches of Beard, who concluded from a study of the primitive sex cells in elasmobranchs that the germinal or sex cells are formed at a much earlier phase in the development of the embryo—the morula or blastula stage—and migrate through the entoderm into the genital area when the coelom is formed. Certain evidence from very early human embryos would appear to support this interpretation. Beard's thesis, then, would derive the seminiferous cells, not from the mesothelium covering the genital ridge, but from primitive sex cells which are budded off the morula and, passing to the genital area, become associated with the mesothelial ingrowths and mesoblastic tissue which provide merely support and nourishment.

In either case the solid anastomosing genital cords enclosing the scanty spermatogonia ultimately acquire a lumen and constitute the early seminiferous tubules. Whether the seminiferous epithelium is derived from the mesothelium of the genital ridge or migrates there from the morula remains uncertain.

*Structure of the Fully Developed Seminiferous Tubule.*—The seminiferous cells are enclosed by a definite basement membrane surrounded by a thick hyaline layer, which again is encircled by a layer of connective-tissue cells.

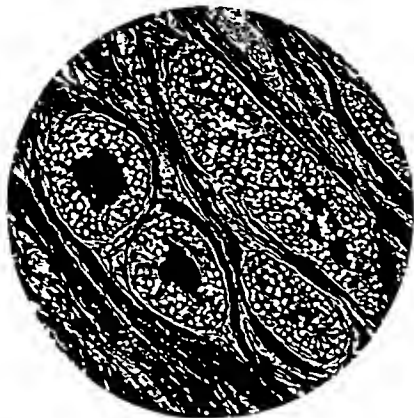


FIG. 197. — Normal testis, showing characters of the seminiferous or germinal cells.

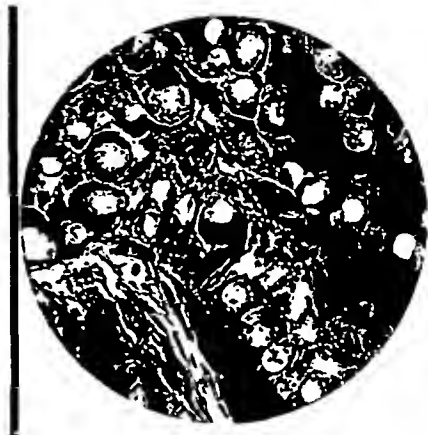


FIG. 198. — Normal seminiferous tubule: high power. The large round cells with clear-cut outline, granular nucleus, and clear protoplasm are spermatocytes.

The germinal cells are arranged in several layers possessing definite characteristics, which are briefly outlined, as having an important bearing on the interpretation of the germinal-celled tumours presently to be described.

1. THE SPERMATOGONIA.—These are rounded clear cells with a large round deeply-staining nucleus, and are set upon the basement membrane. Between them may appear elongated columnar cells which are not always easily made out. These are the sustentacular or nurse cells of Sertoli.

2. THE SPERMATOCYTES.—These are much larger oval or rounded cells disposed in one or more layers situated more centrally. Their nuclei often show various stages of active division.

3. THE SPERMATIDS OR IMMATURE SPERMATOZOA.—These appear as small protoplasmic cells with spherical nuclei lying still more centrally. The heads of the developing spermatozoa are said to retain some connection with the peripheral elongated nurse cells.

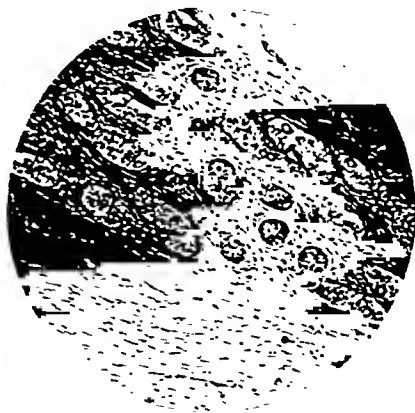


FIG. 199. — Testis from a boy, age 5, showing scanty spermatocytes, large cells with big nucleus and clear protoplasm.

The process of spermatogenesis, then, is as follows: The spermatogonia divide and produce the spermatocytes of the first order. These divide again,



producing the spermatocytes of the second order, which in turn divide and produce the spermatids or future spermatozoa. Cross-sections of the adult tubules show the appearances detailed above, though of course the phases of spermatogenesis vary in different tubules. The important point to note is that the cells are predominantly large and rounded, with a clear protoplasm and a large, deeply staining nucleus, and between the cells there is commonly observed a granular or even reticular framework (*Figs. 197 and 198*). Cross-sections of the immature tubule show chiefly an aggregation of small rounded, oval, or even tapering columnar cells with spherical nuclei (*Fig. 199*), the whole appearance closely resembling that of the early Graafian follicle with enclosed ovum.

**The Excretory Apparatus of the Testis.**—In some respects the testicle presents closer analogies with the kidney than with the ovary; for both in testicle and kidney we are dealing with secreting tubules developed in a special way which have to join up with an excretory apparatus, and in each case defects in fusion between the two sets of tubules have been alleged to account for tumour formations.

The excretory apparatus of the testicle is made up of the straight tubules, rete testis, vasa efferentia, epididymis, and vas deferens. These are all regarded as developed from the Wolffian system, though some doubt obtains about the origin of the straight and rete tubules. There are in addition certain vestigial remnants. The paradidymis, or organ of Giraldès, represents remains of isolated Wolffian tubules, while the hydatid of Morgagni is derived from the abdominal end of the Müllerian duct.

## THE POSSIBLE PATHOGENESIS OF TESTICULAR TUMOURS.

The various tissues of the testis may now be considered from this aspect.

### The Body of the Testis.—

1. THE SEMINIFEROUS CELLS (EPITHELIUM).—We are uncertain as to the precise origin of these cells. Consensus of opinion, however, derives them from the germinal epithelium (mesothelium), and in this case we have the thesis of a specialized epithelium evolved from mesothelial tissues. If a malignant change supervenes, it appears possible that a *carcinoma* may develop from this specialized epithelium, a *sarcoma* from the undifferentiated mesothelium, and from a mixture of these two tissues or from the remarkably close interlocking of the mesothelium and subjacent mesoblast observed in the early stages of development, or from reversionary mesoblastic tendencies, *carcino-sarcomatous combinations* might arise. As a matter of fact carcino-sarcomatous features do occur in certain testicular tumours.

Regarding the seminiferous cells, tumour formations may be assumed to develop in two directions: (a) If the process of spermatogenesis runs wild, a carcinomatous tumour composed of cells of a special type may result. Such a tumour is well designated a *seminoma* or *spermatoeytoma*, and is regarded by some as a common tumour of the testis. If, however, we regard the testis in the same light as any other secreting gland, we should expect simple adenoma of the seminiferous tubules to appear with fair frequency in relation to the malignant adenoma or seminoma, but adenoma testis of this variety

is rarely observed, though, like the small adenoma of the kidney, it may often escape detection. (b) If by any chance the sex cells are stimulated to segment—and in the early stages of development no essential difference can be made out between the sex cells of the ovary and testis—*teratomatous tumours* may result, for these cells, the oögonia and spermatogonia, are regarded as totipotent and capable of giving rise to any or all of the tissues of the body.

2. THE INTERSTITIAL CELLS.—These special and distinctive cells, serving the purpose of providing the internal secretion of the testis, are usually derived from the germinal epithelium, though regarded by some as of simpler connective-tissue origin. The interstitial tissue is often increased in the imperfectly descended testis, and its production can be stimulated by ligature of the vas deferens. In rare cases it may be so abundant as to constitute a definite tumour formation.

3. THE FIBROVASCULAR STROMA.—This is derived from the mesoblast of the genital ridge, and may be expected to give rise occasionally to tumours composed of purely mesoblastic tissue, e.g., fibroma, chondroma, osteoma, lipoma, myoma, endothelioma, sarcoma, and various combinations of these. Metaplasia might be assumed to produce the chondroma and myoma, which are of more particular interest; but as a matter of fact pure connective-tissue tumours are decidedly rare in the testis, though such tissues are common in the teratomata.

**The Straight Tubule.**—Rare tumours called Wolffian carcinomata, to distinguish them from the seminiferous cancers, are related by some to the cubical lining epithelium of the straight tubule, and are considered by others to be of dubious existence.

**The Rete Testis.**—The complicated network of canals called the rete is lined by a flattened or cubical epithelium, supported by a considerable amount of fibrous tissue. The rete is the meeting place where fusion of the secreting and excreting tubules normally occurs, but it is reasonable to suppose that some of the secreting tubules may occasionally fail to join up with the excretory apparatus and that this may initiate in the blind tubules an uncontrolled process allied to spermatogenesis. Mixed elements or 'rests' are not infrequent in this area in early life; lastly, it is a significant fact that new growths often begin in or near the rete, as Curling<sup>1</sup> showed by a process of exclusion.

**Epididymis and Vasa Efferentia.**—Primary malignant disease of the epididymis is of extreme rarity, which appears to point to a relative immunity of the Wolffian derivatives.

To summarize from my observations on a comparatively large number of testicular tumours, there appear to be two clear-cut types, both developing from the reproductive cells themselves.

The first group—the *teratoid*—has been considered with many of its variations in a previous paper. It most probably arises from the segmentation of an aberrant germinal cell, incited by some stimulus at present imperfectly understood.

The second group—the *germinal-celled* tumour—arises from an uncontrolled

proliferation of the seminiferous cells forming the glandular system of the testis in precisely the same way as carcinoma of other glandular organs.

Chevassu,<sup>2</sup> who formulated his classification of testicular tumours on the structural basis detailed above, found that the germinal-celled type, for which he coined the name of *séminome*, and the teratoid variety appeared in almost equal proportions (59–61) in his series of 128 tumours. In my series the proportion has been approximately two to one in favour of the teratoid group, though it is probable that Chevassu's larger series yields a truer estimate of the relative frequency of the two types.

Other varieties of neoplasm can, I think, certainly be regarded as extremely rare, though simple or malignant tumours of connective-tissue origin may be expected to crop up from time to time in any large series. One of my specimens appeared to be a mixed-celled sarcoma developing from the stroma of the testis. No definite example has appeared of lympho-sarcoma testis, which presents several interesting and peculiar features, but I am inclined to think that some of the germinal-celled tumours may readily be mistaken for it.

The germinal-celled group now falls to be considered. Only a few specimens are described, and these are lavishly illustrated by microphotographs with the object of providing a conclusive demonstration of the origin of the germinal or seminal-celled tumour from the seminiferous epithelium.

### THE SPERMATOCYTOMA GROUP.

At first sight it would appear entirely reasonable that the normal process of spermatogenesis should occasionally run riot, so to speak, and give rise to tumours composed of germinal cells and aptly designated spermatocytoma or seminoma, for we have to do with an organ in which cell multiplication proceeds on an enormous scale throughout the greater part of life, and it would seem highly probable that some factor—be it trauma or what not—may occasionally incite a blastomatous process and divert the normal spermatogenesis into a malignant channel. The problem, however, is not so simple as it may appear, and the capacity of the highly specialized cells lining the seminiferous tubules to give rise to a characteristic type of carcinoma called spermatocytoma or seminoma has been alike affirmed and denied by various observers.

Birch-Hirschfeld as far back at 1878 claimed to have traced all stages in the transition from the normal seminal tubules to the malignant phase. The extensive researches of Chevassu, published in 1906, focused attention on this tumour. Chevassu's work appears to have influenced French opinion, and Debernardi<sup>3</sup> and others followed his lead, the controversy then turning in the direction of settling the origin of the tumour from the cells of fully developed seminal tubules, or from unutilized 'rests' which have failed to unite with the seminal system (Pilliet's thesis). Nicholson<sup>4</sup> (1907) accepts a spermatogenic origin for the carcinoma group of his large series, and recent papers indicate that the spermatocytoma has received wide recognition. Tanner<sup>5</sup> records 62 per cent of his collected series of 101 testicular tumours as spermatocytoma, while Southam and Linnell<sup>6</sup> are recent whole-hearted advocates.

Ewing,<sup>7</sup> on the other hand, describes this group as embryonal carcinoma arising from a one-sided development of a teratoid tumour, and raises the following objections to the spermatogenic origin advocated by Chevassu and others :—

1. The characteristic structure is often seen in teratomata—adult or embryonal.

2. In a very early embryonal carcinoma, minute traces of cartilage, entodermal alveoli, and squamous epithelial cell groups were present.

3. The rapid growth of the malignant element gives unusually favourable opportunity for the overgrowth and suppression of other elements.

4. The only known tumour of the adult spermatoblasts (the adenoma testis of Chevassu and others) is very different from the embryonal carcinoma.



FIG. 200.—Seminoma or spermatocytoma, same power as Fig. 198, showing the remarkable similarity between the tumour cells and the spermatocytes in that figure.

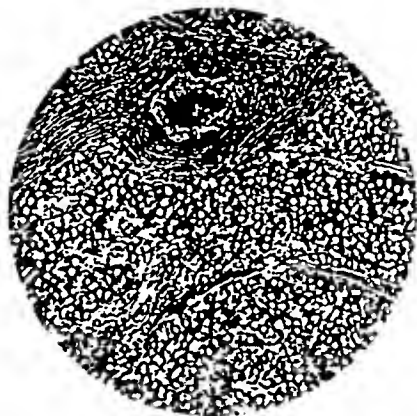


FIG. 201.—Seminoma or spermatocytoma, same power as Fig. 199, showing the striking resemblance between the type of cell in this tumour and the spermatocytes in that figure.

In a previous communication on teratoma testis reference has been made to an undifferentiated round-celled embryonal carcinoma arising from the glandular epithelial elements in certain teratomata and appearing in patchy distribution. Should a one-sided overgrowth take place along these lines and by suppression of the other elements transform the tumour into a pure or almost pure carcinoma, the histological appearances might closely resemble those of the germinal tumour presently described. One would expect, however, such an embryonal cancer to progress rapidly in common with other teratoid cancers, and, as will be shown presently, the germinal tumours are often characterized by a comparatively slow growth.

Several factors have convinced the writer that a specific tumour which may be called seminoma or spermatocytoma, to indicate its origin from spermatogenic or germinal cells, exists beyond all shadow of doubt, though the investigation of this collection of testicular new growths was begun with

rather a bias towards the teratoid theory. These factors may be briefly enumerated :—

1. It is possible to trace transitions between the normal or almost normal seminiferous tubules and the frankly malignant tissue. The evidence backing this statement is submitted in a series of microphotographs (*Figs. 204 to 221*), and if accepted nothing further need be said.

2. The cells of certain tumours are so specific in type that they must have a common origin, and by a process of elimination this origin is best fulfilled by the characteristic cells of the seminiferous tubules (*Figs. 197 to 201*).

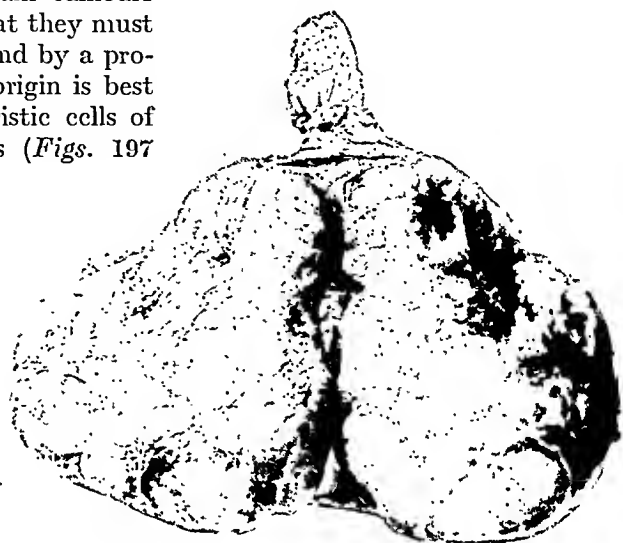


FIG. 202.—Seminoma of three years' duration. The cut surface displays the solid fleshy look so often seen in these tumours. The pale areas microscopically are undergoing degeneration, but have the same structure as the rest of the tumour.

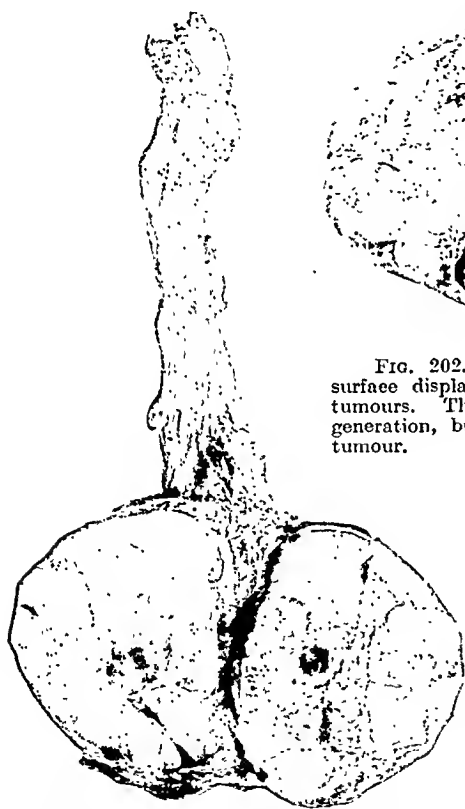


FIG. 203.—Seminoma of two years' duration. The cut surface is lobulated, solid, and fleshy, and lobulation is evident at the periphery. The epididymis, even after two years, is not incorporated.

3. The macroscopic appearances of many, but not all tumours of this variety, suggest that they belong to a specific type (*Figs. 202, 203*).

4. In two teratomatous tumours the cells of the peripheral isolated seminiferous tubules showed signs of proliferation and infiltration, and suggested the possibilities in this direction.

5. The combination of carcinomatous and sarcomatous features in some testicular tumours is more than coincidence, and the explanation must be sought in the early developmental phases of the secreting elements of the testis when mesothelial tissue begins to evolve in an epithelial direction, the carcino-sarcoma being a mixture of these acquired epithelial and reactionary mesoblastic characters. This matter will be discussed later.

*Specimen 1.*—(Labelled round-celled sarcoma.) The tumour was removed from a man, age 47, who had noticed a swelling of the testicle for nearly two years.

**MACROSCOPIC.**—The tumour is the size of the closed fist. The cut surface is firm, fleshy, and homogeneous, and is divided into lobules by septa. The epididymis is not invaded.

**THE MICROSCOPIC FEATURES** (*Figs. 204, 205, 206, 207*).—These may be described under the following heads:—

1. *The Character of the Cells.*—The alveoli are lined by round clear-cut cells with a large round deeply stained nucleus surrounded by a considerable amount of clearer protoplasm. These cells may appear in several layers and form the greater part of the cellular content of the alveolar groups, resembling to a marked degree the peripheral layer of germinal cells of the normal seminiferous tubules. There is, in addition, another type of cell, less numerous but more striking owing to its size. These are much larger rounded or ovoid cells, which lie more centrally and appear to have some connection at their bases with the smaller peripheral round cells. Their nuclei are of great size, often show a nuclear reticulum, and active division is going on apace—in short, they recall at once the large spermatocytes of the normal seminiferous tubules, and together with the other cells the whole appearance suggests irresistibly an uncontrolled process closely allied to spermatogenesis. In certain areas the smaller cells predominate and assume an oval or spindle shape. Many of the cells are strongly eosinophil.

2. *The Arrangement of the Cells.*—A definite alveolar arrangement is readily made out and persists in most parts of the section, though in places irregular cellular patches occur as the result of destruction of the alveolar walls.

3. *The Type of Blood-vessels.*—The blood-vessels in the main are well formed and are typical enough of spermatocytoma as usually depicted, or of carcinoma; but here and there they are of the more embryonic type associated with sarcoma, though confined to the scanty stroma and not appearing between the individual cells.

4. *The Remains of the Testis.*—Testicular substance can be identified as scanty seminiferous tubules located near the upper pole in a portion of the section separated from the rest by an indefinite capsule. Some of these tubules show active proliferation (spermatogenesis) of a fairly normal type, recalling strongly the features of the neoplastic tissue described above, but, what is more significant, a few tubules contain clumps of cells indistinguishable from those of the malignant tissue. If the appearances just detailed are correctly interpreted, then the tumour is certainly a spermatocytoma, and these isolated tubules form the link between the normal and the malignant. A few degenerated tubules also persist at the lower pole.

5. *Other Elements.*—None appear in the various sections indicative of a tridermal origin.

Now let us consider alternative diagnoses:—

1. *The Cellular and Vascular Characters.*—The cells as described may be interpreted as evidence of a sarcomatous process. Indeed, there is little doubt that at first sight the tumour would be regarded by many as a large round-celled sarcoma exhibiting an alveolar arrangement. The blood-vessels are in the main too well formed for sarcoma, and alternatively it stands interpretation as an embryonal carcinoma, thus providing an illustration of the difficulty of differentiating between sarcoma and carcinoma testis.

2. *The Remains of the Testis.*—The fact that testicular substance is present at the upper pole of the testis and is separated from the main mass by an indefinite capsule may be variously regarded. It may represent the remains of the testis which has been isolated by the overgrowth of an encapsuled tumour, as is so common with teratomata. The tumour, then, is most likely to be an embryonal carcinoma of teratoid origin, but at the same time it is possible in a compartmented organ like the testis, which is divided into a number of lobules by fibrous septa, for a carcinoma, sarcoma, or spermatocytoma to involve only a part of the gland, and as the tumour progresses it may crowd one sector to the periphery and lead to an appearance of encapsulation by a condensation of fibrous tissue which really represents a fibrous partition.

*Comment.*—The balance of evidence is in favour of spermatocytoma arising from an exaggerated process allied to spermatogenesis, and the testicular substance near the upper pole provides the link in the conversion of the seminiferous tubules into the malignant germinal tissue.

*Specimen 2.*—(Labelled round-celled sarcoma.) The tumour was removed from a man, age 52, who for three years had noticed that the right testicle was swollen and hard, though not painful till the last two months.

*MICROSCOPIC.*—The specimen is bisected, is roughly pear-shaped, and the size of the closed fist. The cut surface is firm and fleshy, with scattered paler patches and occasional small hæmorrhages. The epididymis is incorporated and indistinguishable. A small piece of granular testicular substance persists at the lower pole.

*Microscopic (Figs. 208, 209, 210).*—

The structure in the main is that of an alveolar carcinoma, though there are abundant encephaloid areas. The cells are of the germinal type, rounded with clear cytoplasm and a large round deeply-staining nucleus. In some areas the alveoli are small and resemble cross-sections of the seminiferous tubules, and in



FIG. 208.—*Specimen 2.* Seminiferous tubule from remains of testis at lower pole showing proliferation of the germinal cells and invasion of stroma.

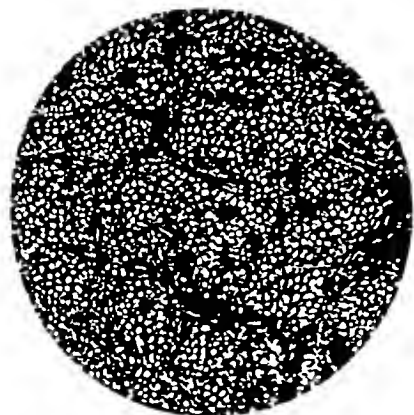


FIG. 209.—Same tumour. Alveolar formation still present though the walls of the tubules have mostly disappeared. Tubules cut transversely.

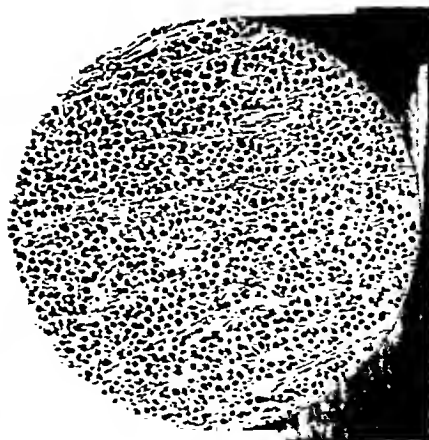


FIG. 210.—Same tumour. Tubules cut mostly longitudinally, the germinal cells appearing as broad sheets with scanty stroma. The tumour cells in Figs. 209 and 210 appear identical with those in Fig. 208.

other parts take the form of long parallel columns, not unlike adrenal cortex, these appearances depending on whether the tubules have been cut transversely or longitudinally.

mostly round or pyriform, but here and there are definitely spindle. In some areas the structure resembles adenocarcinoma and provides an intermediate phase between the typical seminoma and the papillary adenocarcinoma described in the previous specimen.

The early stages in the formation of a lymphoid stroma are well exhibited, the lymphocytes appearing along the line of the vessels in the supporting stroma. The testicular substance present at the lower pole is partly atrophied, but some of the seminiferous tubules show highly active proliferation.

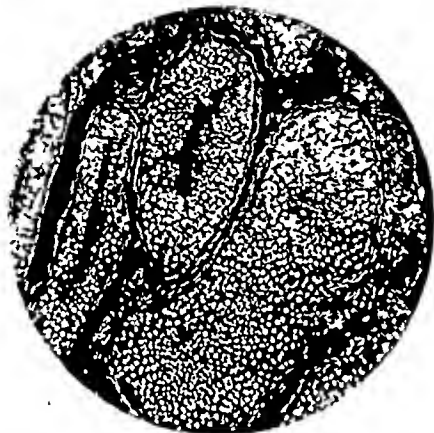


FIG. 215.—*Specimen 4.* Section taken from remains of testis persisting at lower pole, showing seminiferous tubules. The germinal cells are proliferating, and in the largest tubule are beginning to burst through the thinned capsule.

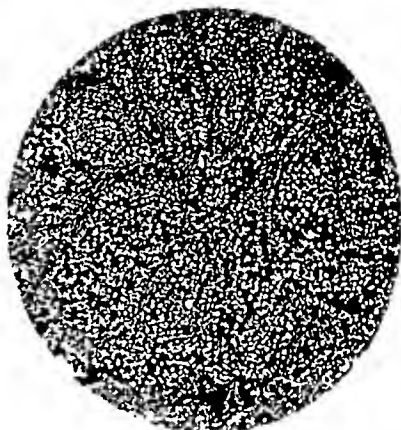


FIG. 216.—Same tumour. The germinal cells retain a well-marked alveolar formation. Along the line of the vessels in the stroma lymphocytes are beginning to appear—the forerunner of the lymphoid stroma seen in some other seminomata.

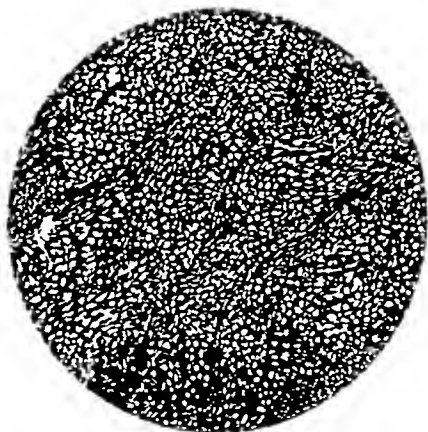


FIG. 217.—Same tumour, showing mixture of round and spindle cells.

*Comment.*—The tumour is a seminoma, but presents variations from the typical round-celled structure. It is noteworthy that this is the only tumour in the series labelled carcinoma, but parts might well stand interpretation as sarcomatous.



*Sub-group : CARCINO-SARCOMA.*

I propose now to describe a sub-group of germinal tumours under the heading of carcino-sarcoma. These are essentially germinal-celled tumours ; but as there is a tendency in some quarters to ignore the sarcomatous potentialities of certain testicular tumours, it seems necessary to state emphatically that combined carcino-sarcomatous features have been observed in a definite percentage of the present series. This peculiar feature as observed in the germinal-celled tumours should not be confounded with carcino-sarcomatous features sometimes seen in the teratoid tumour group, where epithelial and mesoblastic elements may proliferate concurrently and each element contributes to the mixture of malignancy. In the germinal-celled tumour the carcino-sarcomatous potentialities are inherent in the germinal cells themselves, and the connective-tissue stroma takes no part in the formation of the combined tumour.

It is a striking fact in the literature of 'medullary' testicular tumours that doubt often arises as to whether a particular tumour is a carcinoma or sarcoma. So acute an observer as Billroth said that he was often in doubt as to the precise nature of the medullary tumours. This difficulty is a real one and has coloured much of the literature, individual bias accounting for the conflicting statements as to the relative frequency of the two groups. Turning to recent papers, Tanner found 62 per cent of 101 tumours to be germinal carcinoma, while Bulkeley<sup>3</sup> regarded 40 out of his 59 cases as sarcomatous. Nicholson is much nearer the mark with 25 carcinomata (germinal) to 7 sarcomata (or 8, if including one beginning in the epididymis). I would draw attention to the fact that all the germinal tumours described and figured in this series, with one exception, have come to me from a variety of sources with the attached diagnosis of round-celled sarcoma, and it is impossible not to assume that there must be some sound reason for such unanimity.

**Definition of Carcino-sarcoma.**—This term is applied to certain tumours in which carcinomatous and sarcomatous features are blended. The *cells* in most are of an epithelioid character, but sometimes are of the large round, less often small round, oval, or coarsely spindle type so suggestive of sarcoma. In some cases an appearance of a firm granular or reticular matrix can be seen between the individual cells, even when these are typically epithelial, but it should be noted that this appearance is often seen in the cells of the seminiferous tubules. The *blood-vessels* may be of a mixed type in the same tumour. Well-formed or even thickened vessels often predominate, but may be associated in other parts of the growth with vessels of an embryonic type formed of a thin layer of endothelial cells set end-to-end, though the vascular chinks appearing between the individual tumour cells so characteristic of sarcoma are not often seen.

It is not uncommon to see in definitely carcinomatous tumours a patchy degeneration, areas of well nourished, deeply staining neoplastic cells alternating with degenerated, granular, poorly staining tissue. These appearances suggest that the tissue furthest removed from the vascular supply degenerates from deficient nutrition, for the deeply stained tissue is often aggregated in the form of nodules around thin-walled vessels. This appearance has been

described as angio-sarcomatous, but certainly occurs in undoubted carcinoma. In other tumours of the papillary adenocarcinomatous type a spurious peritheliomatous appearance may be produced by cross-section of the papillae if they possess a highly vascular core.

The most reasonable explanation of these combined characters as observed in the germinal tumours is supplied by the *mesothelial* origin of the secreting elements of the testis. If we turn, for example, to the adrenal we find a parallel, for the adrenal cortex is developed from mesothelium. Certain cortical adrenal neoplasms may display sarcomatous features, though in the main the acquired epithelial characters predominate. Adami<sup>9</sup> has coined the term 'mesothelioma' for tumours derived from mesothelial structures, and as such structures play a dominant rôle in the development of the body of the testis we may reasonably expect to find some tumours displaying combined carcino-sarcomatous features, although, as in the adrenal, the acquired epithelial characters usually predominate.

So far as the evidence of metastasis goes, some tumours probably spread both by the blood- and lymph-streams. In two personally observed cases of carcinoma exhibiting partial sarcomatous features, metastasis involved both the lumbar glands and the liver. The majority of cases, however, behave as cancers, and metastasis usually first involves the lumbar lymphatic glands, though it is interesting to note that the traditional sarcoma testis, like round-celled sarcoma (?) of the thyroid, has in the past been credited with a special tendency to spread by the lymphatics.

The following two specimens are described as examples of carcino-sarcoma. They are essentially germinal-celled tumours in which sarcomatous characters are accentuated, and differ only in degree from the tumours previously described :—



FIG. 218.—*Specimen 5.* Shows recognizable seminiferous tubules persisting in one area of the tumour.

*Specimen 5.*—(Labelled round-celled sarcoma.) The tumour was removed from a man, age 46, who for three years had noticed gradual enlargement of the testicle. The inguinal glands on the same side were involved, and there was also a mass in the lumbar region. Castration was performed to relieve the patient of the pain and discomfort caused by the weight of the tumour.

**MICROSCOPIC.**—The specimen is bisected and is roughly the size of an ostrich's egg. The cut surface is distinctly lobulated. The greater part of the tissue in the lower two-thirds has the appearance of being encapsulated, is split into lobules by fibrous septa, and shows hæmorrhagic infiltration and several patches of softening and others of pale degenerated tissue. The tissue forming the upper third is firm and more homogeneous. The epididymis is fused with the tumour.

**MICROSCOPIC (Figs. 218, 219, 220).**—The structure is that of a germinal carcinoma, but certain areas are so distinctly sarcomatous in type as to justify the

existing diagnosis. The cancerous areas appear both in the scirrhus and encephaloid form, and the cells are definitely germinal in type.

*Sarcomatous features* are evidenced by (a) round-celled characters—spindle in some areas, (b) embryonic blood-vessels and hæmorrhagic infiltration, (c) inter-cellular matrix. The inguinal glands show marked invasion, also of mixed character.



FIG. 219.—Same specimen, showing round-celled structure and thin-walled vessel.



FIG. 220.—Same specimen, showing mixture of round and spindle cells.

*Comment.*—This particular tumour, derived undoubtedly from germinal cells, exhibits a structure which may with justification be described as carcinomatous or sarcomatous according to individual bias. I regard it as a carcinoma arising from germinal cells, but exhibiting marked sarcomatous tendencies. Such a tumour can only arise from cells which possess both epithelial and mesoblastic potentialities, and the mesothelial tissue from which the secreting cells of the testis develops is the only likely source.

There is *metastatic invasion* both of the inguinal and lumbar glands, which serves to indicate the predominantly carcinomatous character of the tumour.

*Specimen 6.*—(Labelled round-celled sarcoma.) The specimen comprising the testicular tumour, spermatic cord, and associated mass of lumbar glands was obtained post mortem, and the age of the patient is not stated. In addition to the lumbar metastasis there were also secondary deposits in the liver.

*Macroscopic.*—The testicular tumour resembles the previous specimen in

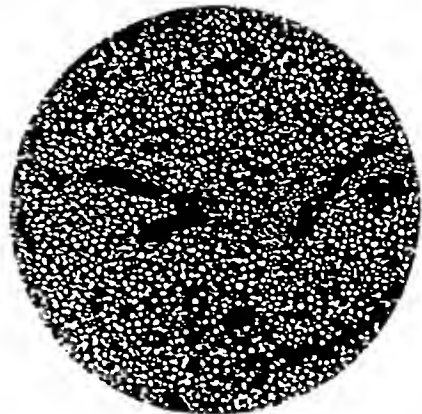


FIG. 221.—Specimen 6. Shows round-celled structure and capillaries lined by thin spindle cells. The tumour is old, and the cells are not so large as in the previous recent specimen.

size, and, as in it, the cut surface is divided into a lower lobulated area undergoing cystic degeneration and an upper solid area. The epididymis is incorporated.

**MICROSCOPIC** (*Fig. 221*).—

**Testis.**—The structure is that of the specific germinal tumour and is partly alveolar and partly encephaloid. Parts of the tumour are decidedly sarcomatous, if importance is to be attached to embryonic blood-vessels and to spindle-celled areas.

**Lumbar Glands.**—These are almost entirely replaced by neoplastic tissue displaying the same mixed characters. The hepatic deposits are not available.

**Comment.**—The tumour is of the germinal-celled variety, but, as with the previous specimen, there is good reason for the existing diagnosis. The metastases in the liver possibly stand interpretation as evidencing a blood-spread, in which case both elements of the tumour have produced metastases.

### GENERAL CONSIDERATIONS.

Certain points arise for consideration out of the preceding examples, always remembering the danger of drawing inferences from comparatively few cases.

**1. The Age of the Patient.**—With only one exception these data are available, and the ages range from 39 to 52 years, with an average of 45. Nicholson, who accepted a germinal origin for his 25 carcinomata, found the average age incidence to be 43 years, and Schultz and Eisendrath<sup>10</sup> give it as 42.6 for the spermatocytoma group. These figures correspond closely, and we are justified in assuming that the specific germinal tumour develops most commonly in the forties, or in middle life, just when proliferative changes in, e.g., the mammary epithelium manifest themselves. At the same time it should be remembered that carcinoma of teratoid origin is also apt to declare itself in the same decade.

**2. The Duration of the Tumour.**—Reference to the quoted histories makes it clear that enlargement of the testicle had been noticed for a comparatively long time—three years in two cases and two years in two others. For what it is worth the inference here seems obvious that the specific germinal tumour is apt to be slow-growing. In three years *Specimen 2* had only reached the size of the closed fist, and in two years *Specimen 3* had only reached the size of an orange.

The teratoid carcinoma, on the other hand, arising in the fourth (or any other) decade, advances much more rapidly. Two examples will suffice to illustrate this point. The first reached the size of a small melon in six months and had already invaded the inguinal glands. The second attained the size of the closed fist in four months and had already fungated through the scrotum and invaded the inguinal glands. In each case the epididymis was fused and indistinguishable.

**3. Involvement of the Epididymis.**—French writers, Jeanbrau<sup>11</sup> for example, state that the epididymis long remains free in spermatocytoma. This is certainly true of the smaller examples (*Specimens 3 and 4*), but when a tumour is of long duration and has attained a large size it usually invades and incorporates the epididymis. Whether the epididymis remains free or not seems to depend partly on the site of origin of the tumour process in the body of the testis, and partly on its duration. If the blastomatous process

begins in or near the rete, as is usually stated, it naturally would be likely soon to infiltrate the adjacent epididymis, but with a more central origin the epididymis would probably escape till late in the disease (*Specimen 2*, of three years' duration).

**4. Macroscopic Appearances.**—The germinal tumours are typically solid, and the cut surface firm, fleshy, and homogeneous except for occasional whitish areas indicating patchy degeneration. The cut surface is often traversed by fibrous septa producing distinct lobulation, and sometimes imparting lobulation to the surface of the tumour. Occasionally softening and cystic degeneration may be present, but they are late developments.

**5. Microscopic Appearances.**—These may be considered under the following heads:—

*a. Characters and Variations of Cells.*—It is reasonable to expect that the influence of the blastomatous process may produce variations from the absolutely typical seminiferous cell, and this probably accounts for the fact that some germinal tumours are predominantly composed of large cells with a clear cytoplasm of the spermatocyte type, while others are composed of smaller cells with large darkly staining nuclei and much less cytoplasm, while in others again the cells may appear in parts of a remarkably large, rounded, polygonal, or pyriform type. The mesothelial origin of the germinal cell supplies sound reason for individual variations, and I am inclined to believe that reversionary tendencies play a large part in the production of the so-called lymphosarcoma testis. All through this group, as described previously, it is impossible to ignore the fact that we are dealing with cells which in some areas of the same tumour may be predominantly epithelial and in others sarcomatous, and if we allow full scope to these two potentialities we may reasonably expect, and do find, tumours ranging from adenoma or adenopapillary carcinoma down to others composed of small round cells hardly, if at all, distinguishable from lymphosarcoma, yet all arising from germinal cells.

The germinal cells, when lying loosely as seen in thin sections, are clear cut and free from any intercellular matrix. When closely packed, as in encephaloid patches, the cells tend to become polyhedral from mutual pressure and assume a mosaic appearance, and a fine granular or even reticular matrix appears between them. This appearance, however, is commonly observed in normal seminiferous tubules, and I am not inclined to attach much importance to it as evidencing sarcomatous tendencies, but regard it as a degeneration product thrown out from the cells under pressure.

It is an interesting feature in some of these tumours that many of the cells are strongly eosinophil. Southam and Linnell mention this point as supporting carcinoma, and it has been observed in several of the present series. It may be noted that in some control sections of normal testes the cells of the seminiferous tubules have been noticeably acidophil.

*b. Arrangement of Cells.*—In the early stages of the tumour, and sometimes even in the advanced, the tubular or alveolar arrangement is often preserved, so that portions of the spermatocytoma may differ from normal testicular substance only in a greater germinal-cell content and a corresponding reduction in the supporting stroma. Depending on the way in which the

tubules are cut, a markedly alveolar arrangement may predominate or the germinal cells may appear in closely-packed parallel sheets, separated by vessels enclosed in a scanty stroma, reminiscent of the adrenal cortex. In advanced stages and in rapidly growing tumours all alveolar arrangement may be lost except in scanty patches, and a predominantly encephaloid appearance results.

*c. Retrogressive Tendencies.*—These are occasionally observed in areas of these tumours. The connective-tissue stroma appears to gain the upper hand, and as it grows strangles the germinal cells, which become smaller, irregular, and granular.

*d. Type of Blood-vessels.*—It is stated that the blood-vessels in the specific germinal tumour are well formed and typical of carcinoma generally. This is true up to a point, but does not go far enough. In early cases the vessels are well formed in the main, but it is nearly always possible by repeated sections to find areas in which they are embryonal in type, and in other cases they may be typically sarcomatous. Consequently, too much stress should not be laid on the blood-vessel type in the diagnosis of seminoma or spermatocytoma, and, furthermore, these vascular peculiarities are likely to lead one astray in the interpretation of the small round-celled type of tumour resembling lymphosarcoma.

*e. Lymphoid Stroma.*—Chevassu describes a lymphoid stroma as a feature of the seminoma, and Ewing of embryonal carcinoma. All stages of its formation may be traced in the present group. In the beginning the lymphocytes appear along the lines of the vessels in the scanty stroma, and gradually accumulate and proliferate as the stroma and contained vessels disappear from the picture. Ultimately the lymphocytes come to form an interlacing network enclosing individual alveoli or groups of alveoli. This peculiar condition, I think, is best regarded as a connective-tissue reaction which seeks to confine the activities of the tumour cells—on the same lines, for example, as the round-celled barrier opposing the downward growth of the epithelial cells in a cancer of the lip or tongue.

In addition to, or apart from, a scanty or abundant lymphoid stroma, it is not uncommon to see nodules of lymph corpuscles scattered irregularly through the tumour tissue.

**Heterologous Elements.**—None appear in the various sections examined from the tumours of this group, but it must be freely admitted in fairness to the teratoid theory that an elaborate examination to exclude their possible existence has not been attempted. At the same time my experience has been that with the teratoid group piecemeal examination of different areas of a tumour invariably reveals its teratoid nature. Allowing for the imperfections in the methods of examination, it seems reasonable to conclude that those tumours are homologous, and arise from the germinal cells of the seminiferous tubules.

**Choice of a Name.**—The term ‘spermatocytoma’ which has recently come to be widely applied to these tumours of germinal-cell origin is open to objection. It implies a tumour composed essentially of cells resembling spermatocytes, i.e., large rounded cells with a big darkly staining nucleus and a considerable amount of clear cytoplasm. Some germinal tumours fulfil this

condition, but others do not and are composed of much smaller cells, and in others again the cells may assume giant proportions. All these are more probably variations from the germinal cell arising at some phase of the sexual cycle.

Chevassu's choice of term, 'séminome', already anglicized to 'seminoma', is preferable, because it only implies an origin from seminal cells and leaves more latitude. In many respects these tumours would be most accurately described under the heading of 'mesothelioma' to indicate their fundamental origin and their frequently combined carcinomatous and sarcomatous characters, but this term is not likely to gain popular acceptance. Short of it, seminoma is the best and most comprehensive term, and allows scope for individual cell variations.

### CONCLUSIONS.

1. The seminoma or germinal-celled tumour is one of the two common neoplasms of the testis, the other being the teratoma and its many variations, discussed in a previous communication.

2. *Origin*.—It arises from a proliferation of the cells of the seminiferous tubules in precisely the same way as glandular carcinoma of other organs.

3. *Age Incidence*.—It declares itself most frequently in the fourth decade.

4. *Rate of Growth*.—It advances more slowly than teratoid carcinoma.

5. *Structure*.—It presents in the main a specific structure which may be definitely related to the seminiferous cells, but is subject to certain variations in type.

6. *Carcinoma or Sarcoma?*—Carcinomatous features usually prevail, but it is common to find mixed histological characters in the same tumour. This feature is probably related to the mesothelial origin of the seminal cells and to reversionary tendencies.

7. *Metastases*.—Most germinal-celled tumours behave like cancer and spread by the lymphatics. Occasional spread by the blood-stream is to be related to sarcomatous characters.

The photographic work has been done by Mr. Frank Pettigrew, Technical Assistant to the Department of Surgery, to whom I am greatly indebted for his skill and care.

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## LYMPHATICOSTOMY FOR PERITONITIS.\*

By C. HAMILTON WHITEFORD, PLYMOUTH.

LYMPHATICOSTOMY is the operation of exposing and draining the cervical portion of the left thoracic duct. The operation aims at diverting from the blood-stream bacteria and toxins absorbed from inflamed peritoneum and intestines, and was performed for the first time for peritonitis by Costain, of Toronto, in October, 1922.

### ANATOMY OF THE THORACIC DUCT.

The anatomy of the cervical portion of the duct varies greatly. Wylls Andrews<sup>1</sup> states that the duct may be double or even triple. The right duct may be equal to, or more developed than, the left. The duct may enter the subclavian vein by two or three branches, or may enter a vein other than the subclavian. Regarding the relative frequency of these variations, Wylls Andrews<sup>2</sup> quotes the results of several series of dissections. In one series of 40 cases, in 18 the duct had two branches, and in 9 of these 18 the duct re-united; 7 had two insertions into the vein, and 2 had four insertions. In another series of 24, the duct was branched in 6. In a third series of 17, the duct was branched in 8. Gerrish<sup>3</sup> considers that "The size of its terminal portion depends upon the presence or absence of branches given off to the veins as it rises through the thorax".

Costain<sup>15</sup> supplies the following details: "There is a suction force on the thoracic duct, due to the venous blood passing the opening of the duct, similar to that created by the Sprengel pump. In lymphaticostomy the ligature cuts off this suction, and when the duct is opened there is a flow of lymph. The duct does not become distended when ligated, and the lymph-flow is less than one would expect from observing the flow through the duct before ligaturing and opening. [If this suction is the chief cause of the lymph entering the vein, the profuse discharge, recorded in cases of lymphaticostomy with ligation of the duct, ought not to occur. Probably the respiratory movements are an equally important factor.—C. H. W.] The normal duct is transparent, its contents being visible. In peritonitis, however, the fluid may be bloody, and the duct may resemble a vein, and must then be distinguished by its position."

The difficulty of finding the duct in the cadaver is due to the fact that the duct is collapsed, and in that condition resembles fibrous tissue. It is more difficult to find in the dog than in man.

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\* A paper read before the Plymouth Medical Society, March 17, 1925.



### EXPERIMENTS.

Costain<sup>1</sup> produced in dogs a septic peritonitis which, when untreated, proved fatal in about forty-eight hours. Ligation and drainage of the thoracic duct performed during the first twenty-four hours (without drainage of the peritoneal cavity) saved many of the dogs.

Williamson and Brown,<sup>5</sup> in the Mayo Clinic, carried out experiments on dogs in order to decide whether passage of bacteria from the abdomen to the lungs, via the thoracic duct, was a cause of pneumonia following abdominal operations. They employed the *Bacillus prodigiosus* as being not normally found in the alimentary canal, non-pathogenic, and easily identified. With the thoracic duct opened, bacteria, either swallowed or injected into the jejunum or into the colon, were never obtained from the duct; but bacteria injected into the peritoneal cavity appeared in lymph from the duct two to four hours after injection, in 4 out of 8 cases.

### CLINICAL CASES.

In conditions other than peritonitis the duct has been opened in the neck: (1) For fat embolism by Wilms<sup>6</sup>; the divided duct drained freely and closed after six days; the patient recovered. (2) In the course of operations in the supraclavicular area; Fredet<sup>7</sup> collected 58 such cases, and of these only 5 died; the majority of the cases, when the fistula did not close spontaneously, were treated either by ligation of the duct or by compression. (3) By accidental (non-operative) injury. (4) In cases of abdominal cancer (Jonnesco<sup>8</sup>).

Recorded cases of human peritonitis treated by lymphaticostomy are few in number, and being widely scattered throughout surgical literature are difficult to trace. The following list of such cases has been collected, but makes no claim to completeness.

*Case 1 (Costain<sup>2</sup>).—*Pneumococcal peritonitis, in a girl, age 9. The abdomen was not operated upon, but the patient was exsanguinated and transfused. Pneumococci (Type IV) were found in pus aspirated from the abdomen. No bacteria were found in the blood or in the lymph, which was not tested for toxicity. Toxic symptoms rapidly disappeared. Drainage from the duct, at first free, ceased on the seventh day. On the twentieth day after operation the patient was discharged cured.

*Costain's Technique.*—A three-inch incision is made along the lower posterior border of the left sternomastoid muscle. Blunt dissection exposes the sternomastoid muscle, which is drawn forwards, and the omohyoid muscle, which is drawn upwards and backwards. The internal jugular vein, exposed down to its junction with the subclavian vein, is rolled forwards, exposing the thoracic duct. The duct is ligated with catgut close to its termination, and a half-inch incision is made in the long axis of the duct above the ligature. A narrow strand of rubber is passed into the duct. The operation is performed under either general anaesthesia or local infiltration.

Following Costain's lead, other surgeons have performed lymphaticostomy for peritonitis.

*Case 2* (Edwards<sup>10</sup>).—Puerperal pneumococcic peritonitis. The patient, age 17, was operated upon thirty days after confinement, under novocain anæsthesia. On the third day improvement in the abdominal and general condition was marked and sustained. The wound healed by granulation, and the patient was discharged cured fifty-one days after operation. Glucose was given intravenously both before and after operation. Transfusion of citrated blood was performed on the day before operation. Blood cultures were negative, but the fluid from the duct contained pneumococci. Drainage from the duct was free during the first three days, and ceased on the eighth day.

*Case 3* (De Lee<sup>11</sup>).—Puerperal peritonitis and septicæmia in a primipara, age 25. The primary septic focus was in the perineum, whence the infection spread to the peritoneum and blood-stream. Streptococci were found in the blood in pure culture. Under local anæsthesia, the patient being nearly comatose, the duct, which entered the internal jugular vein, was found with some difficulty and was drained. "Within half an hour the patient's mind cleared up, and she sat up and demanded food. In about eight hours, however, coma returned, and she died the next morning". The fluid from the duct was profuse but sterile. No operation was performed upon the abdomen.

*Case 4* (Cooke<sup>12</sup>).—A man, age 48; septic peritonitis, treated by removal of a perforated appendix, the abdomen being closed without drainage. Lymphaticostomy was performed immediately after the cœliotomy. "The duct was ligatured off from the vein, slit up, and stitched to the skin". After a stormy convalescence, the patient was discharged cured, twenty-five days after operation. The duct was hard to identify, being "delicate in texture and thinner than one expects". Beads of lymph were visible in the unopened duct. The patient having been dehydrated by vomiting and starvation, the flow of lymph became free only after twenty-four hours of continuous rectal saline.

*Case 5* (D. A. Mitchell<sup>13</sup>).—A briefly recorded case of "ruptured intestine" of sixty hours' duration, with loss of liver dullness. After lymphaticostomy the patient lived twenty-four hours, during which time the duct drained freely. The difficulty of the operation lay in having to work "practically behind the clavicle".

*Case 6* (Alexander Mitchell<sup>14</sup>).—Pneumococcal peritonitis in a girl, age 4 years. Cœliotomy evacuated a large amount of pus. A drainage tube was passed into the pelvis through the lower angle of the incision. Improvement, local and general, followed, and lasted for a week. Then the abdomen again became distended and painful, with rise of temperature and pulse. Lymphaticostomy was performed fourteen days after the cœliotomy. Lymph drained freely, with general and local improvement for five days. Then, "although the abdominal condition appeared to be most satisfactory, and the child was taking abundant nourishment by the mouth, she seemed to be getting weaker and to be losing weight rapidly". A free flow of lymph continued up to shortly before death, which occurred nine days after the lymphaticostomy. Pneumococci (Type II) were found in the abdominal pus. No organisms were found in the lymph.

Mitchell says: "From observation of this case alone, the impression was that the lymphaticostomy had a remarkably beneficial effect on the course of the peritonitis, but that it seemed highly probable that the continuous discharge of lymph from the thoracic duct was the ultimate determining cause of the patient's death." Also: "The main difficulty seems to me to be, what is going to happen to the patient if his duct fistula does not heal when his abdominal condition improves. If the discharge of lymph continues for more than a few days, it is obvious that the loss of the essential food constituents must be a very serious matter." He asks for Costain's opinion as

to what happens when the fistula ceases to discharge, and whether it is possible that the ligature on the duet is absorbed and circulation through the duet resumed.

*Case 7 (Costain<sup>15</sup>).—*A second case of recovery, in a boy, age 10 years, who, after four days' illness, had cœliotomy performed, disclosing a ruptured appendix and diffuse peritonitis. The appendix was removed, with drainage both of the peritoneum and the terminal ileum. On the day after the cœliotomy, since he was going downhill, lymphaticostomy was performed under gas and oxygen. A strand of silkworm gut was passed down the duet for about 1 in., a rubber tube being threaded over the silkworm gut down to the duet. No mention is made of ligation of the duet close to the vein. No bacteriological findings are given. The flow of lymph was profuse for forty-eight hours and had ceased by the fifth day. The tube and silkworm gut were removed on the second day. Recovery was complete.

Mackenzie<sup>16</sup> has performed lymphaticostomy twice, but does not give details or results. He mentions the technical difficulties in identifying the duet on account of its small size and thinness, and of inserting and retaining the rubber drain in the duet. To overcome the latter difficulty he advocates, but has not tried, a tapering cannula with a bulbous tip which is retained in the duet by a fine catgut ligature. The cannula, being made of silver, can be bent to fit the wound.

The writer has knowledge of two other cases (not yet published) of peritonitis treated by lymphaticostomy. Both died.

### COMMENTS.

In the above collection of 11 cases, details are available in 7, with 3 deaths and 4 recoveries (*Cases 1, 2, 4, 7*). In 3 cases the infection was pneumococcal, and of these, 2 (*Cases 1 and 2*) recovered. In none of the 7 cases is there a record of examination of the lymph for toxicity. Such examination is especially called for when the lymph is found to be sterile. The effect of the lymphaticostomy on the peritonitis is difficult to estimate, especially when, as in some of the cases, the peritonitis has been treated by cœliotomy. But it is to be noted that the peritonitis was not treated by abdominal operation either in Costain's experiments on dogs, or in *Cases 1 and 2*, which recovered. Equally difficult is it to say, in regard to the cases which died (except in *Case 6*), whether, or to what extent, the lymphaticostomy contributed to the fatal termination. Excluding the pneumococcal patients, the cases which died without drainage of the abdomen raise the question whether the omission of drainage did not deprive these patients of a possible chance of survival. The amount and duration of the lymph-flow from a partially or totally divided thoracic duet probably depends largely upon the anatomical arrangement of the lymphatic system.

In any given case of lymphaticostomy, and in cases of fistula following unintentional surgical injury or accidental trauma, it is usually impossible to say whether the duet consists of a single trunk or of several branches, while except in lymphaticostomy it is known only rarely whether the duet has been partly or completely divided.

In cases which survive, with closure of the fistula, there are three possible

routes for the chyle, which may reach the blood-stream by: (1) Other branches existing in the left side of the neck; (2) The right duet or thoracic veins, by enlargement of branches of communication within the thorax; (3) Its original course, the injured duet undergoing repair; and (4) Any combination of these three routes. In regard to route 3, it is known that, throughout the body, small ducts, even when tightly ligated, occasionally manage to re-establish their lumina, but such restoration of the lumen takes time and cannot be relied on. Also, it is a clinical fact that a small duct incised in its long axis has a strong natural tendency to heal without serious stricture, provided there is no complete obstruction of its lumen.

In lymphaticostomy, since there is always the possibility that the duet which is drained may be the chief route for the chyle, a permanent obliteration of the lumen, either by tight ligation or transverse division, involves the risk of depriving the patient of a large amount of nutriment. An operation which (as in *Case 6*) rescues the patient from peritonitis, only to kill him by starvation, is lamentable. Preservation of the lumen of the duet would provide a definite chance of some, at least, of the chyle reaching the blood-stream by its accustomed channel.

It appears desirable to provide during the lymphaticostomy: (1) For preservation of the lumen of the duet; (2) A simple method of stopping, at any given moment and without further operation, the escape of chyle from the fistula. The writer ventures to suggest that these two ends might be attained by the following modification of Costain's technique. For a large duet, employ the cannula suggested by Mackenzie. The larger the duet, the less difficult its intubation and the greater the probability that the duet is the chief route for the chyle. The cannula is advocated, not so much on account of difficulty in inserting the rubber drain (Mackenzie's reason for using the cannula), as because the cannula renders superfluous the ligature on the duet close to the vein. The bulbous tip of the cannula is retained in the duet by a catgut ligature tied as the first half of a surgical knot, the ends being left long. The catgut, hardened to resist absorption, must not be so fine as to cut easily into the duet walls. For removal of the cannula, the knot is loosened and the ligature is left *in situ*, encircling the duet. Should the discharge from the fistula continue for so long a time, or in such an amount, as to endanger the patient's nutrition, the escape of chyle can be stopped by tightening the ligatures. Should the fistula close spontaneously, the ligature can be easily removed. If experience proves that the segment of duet compressed between the ligature and the cannula tends to become necrosed, an extra ligature placed loosely round the duet above the cannula during the lymphaticostomy will provide a means of closing the fistula.

When the duet is too small for intubation, a loop or strand of silkworm gut takes the place of the cannula, and two ligatures similar to that previously described are placed loosely round the duet on each side of the fistula. The distal ligature is tightened only when the flow from the fistula is so slight as to suggest that an appreciable amount of lymph is still flowing past the fistula into the vein. The proximal ligature provides for stopping the flow from the fistula. The ligatures, when no longer required, can be removed by loosening their half-knots.

Proximal ligation of the duct, although stopping the external loss of chyle, does not necessarily imply an *immediate* restoration to the blood-stream of an amount of chyle equal to that which has been escaping from the fistula. The amount of chyle returned *at once* to the circulation depends largely on whether competent accessory lymph-channels already exist or have to be developed. This, in any given case, is an unknown factor. The records of accidental injuries to the duct which healed without ligation suggest that the omission of the ligature on the duct between the fistula and the vein entails only a theoretical risk of air entering the vein via the fistula.

### INDICATIONS FOR LYMPHATICOSTOMY.

Costain<sup>15</sup> says: "The indications for the operation are not yet clear", and that surgeons are not unanimous in accepting the theory that most of the post-operative symptoms regarded as complications are due to septic absorption. He considers the operation to be indicated in:—

1. Secondary peritonitis, as in Cooke's case (*Case 4*), as an adjunct to dealing with the condition locally. In some cases cœliotomy and lymphaticostomy have to be done simultaneously. [In regard to *Case 4*, advocates of drainage will argue, not unfairly, that drainage of the peritoneal cavity might have obviated the lymphaticostomy.—C. H. W.]

2. In all cases in which, after the abdominal operation, symptoms of continued septic absorption persist. Lymphaticostomy should not be delayed until the patient's condition is hopeless.

3. "In primary peritonitis, especially the pneumococæcæ, in which the diagnosis is clearly established." "When the diagnosis is doubtful, the abdomen should be opened first, in order to verify the condition".

Costain says in conclusion: "I believe that, with good judgement in election and skill in the performance, lymphaticostomy will lower the mortality rate in diffuse peritonitis, by saving those to whom relief could not otherwise come".

### CONCLUSIONS.

1. No general agreement has, as yet, been arrived at on the indications for lymphaticostomy.

2. The experimental and clinical evidence at present available suggests that a further trial of lymphaticostomy is desirable.

3. Much more clinical experience of lymphaticostomy must accumulate before the value and risks of the operation can be decided.

4. Identification of the duct may be difficult. In at least 30 per cent of cases (nearly 40 per cent in the combined series quoted by Wyllys Andrews) the operator must be prepared to find a duct which does not consist of a large single trunk opening into the subclavian vein.

5. In recorded cases in which the escaping lymph was sterile, the toxicity of the lymph appears to have been taken for granted. In records of future cases, proof of such toxicity should be given.

6. Permanent or prolonged obliteration of the lumen of the duct, by complete division or by ligature, appears to accentuate unnecessarily the risk

of a flow from the fistula so persistent and profuse that death occurs from starvation. Preservation of the lumen might give the chyle a chance of resuming, in part at least, its original route. Provision should be made during the performance of lymphaticostomy for stopping the loss of chyle without having to perform a further operation on a debilitated patient. A technique which may accomplish this is described.

Since the above was in type, the two following papers have appeared.

H. H. Cox and L. B. Bell<sup>17</sup> experimented with dogs on lines very similar to those of Costain, but failed to confirm Costain's findings. They consider that the current of lymph is maintained by respiration, each inspiration producing within the thorax a temporary negative pressure which withdraws chyle from the abdomen. Fistulous drainage appeared to hasten the death of the animal, probably by starvation. Ligation of the thoracic duct prolonged the lives of the dogs, probably by retarding absorption of toxins. "Suspended carmine particles and methylene blue were injected into the free peritoneal cavity, and in two cases appeared in the chyle from the thoracic duct within five minutes. In two other cases it did not appear within forty-five minutes. The results indicate that the rate of absorption through the thoracic duct is by no means constant."

L. D. McGuire<sup>18</sup> conducted in the Mayo Clinic experiments with *B. prodigiosus* in peritonitic dogs. He found that: (1) *B. prodigiosus* was never found in lymph from the thoracic duct; (2) The toxicity of the lymph from the duct, tested by intravenous injection into rabbits, was practically nil; (3) Peritonitis in dogs was not benefited by lymphaticostomy. McGuire's results differ from those of Williamson and Brown<sup>5</sup> in the Mayo Clinic.

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# LYMPHATICOSTOMY IN PERITONITIS.

By ARTHUR COOKE, CAMBRIDGE.

THE efforts of general surgeons to reduce the mortality from acute general peritonitis have been crowned with so much success during the last twenty-five years that we are apt to put the matter aside as sufficiently dealt with by prompt emergent surgery. It is a platitude to say that success largely depends on early diagnosis, efficient transport, and effective surgery. Where one of these fails to function, from whatever cause, the fate of the inflammatory type, of acute abdomen as distinguished from the obstructive type is death from peritonitis in the majority of cases. No doubt other causes of death, too obvious to mention, play their part, but it will probably be generally conceded that acute general peritonitis is the most frequent.

On consulting the Registrar-General's Report for 1923, published in 1924, the following figures for England and Wales are of interest :—

Deaths from	appendicitis	..	..	2826
"	"	duodenal ulcer	..	907
"	"	gastric ulcer	..	2105
"	"	hernia	..	1738
"	"	biliary calculi	..	1030
"	"	peritonitis (cause unstated)	..	417
"	"	puerperal sepsis	..	985

In a previous year (1920) the deaths from gastric ulcer were classed as 'perforating ulcer of the stomach, 1975'. It is impossible to state with any degree of accuracy what number of deaths in the above list was caused by peritonitis, but a moderate estimate would probably be one half, or about 5000: no excuse is necessary for recording cases which may point to a means of reducing that number.

Costain's<sup>1</sup> records of lymphaticostomy in dogs at the time of tying the appendix with the object of producing general peritonitis are as follows :—

- 7 dogs had appendix and mesentery tied, with no lymphaticostomy: all died in forty-eight hours of peritonitis
- 11 dogs had appendix and mesentery tied, with simultaneous lymphaticostomy: 4 recovered, having developed thoracic duct fistula; 7 died without developing fistula of thoracic duct.
- 7 dogs had appendix and mesentery tied, with lymphaticostomy twenty-four hours later: 3 recovered—all had duct fistula; 3 died with no fistula, there being collateral flow of lymph; 1 died from pneumonia.

Assuming for the moment that lymphaticostomy is advantageous in peritonitis, the difficulty in human surgery is to decide when general peritonitis is of such severity as to demand this further operation in addition to removal

of the cause. To convey outside the body the main stream of lymph which is assumed to carry into the blood a considerable amount of the toxins absorbed from the peritoneum appears desirable. No one would advocate this remedy for a moribund patient; no one would think it necessary for an appendicitis of good prognosis with a moderate peritonitis likely to be controlled by the ordinary methods. Between these two extremes there is a fairly large class known to surgeons engaged in active emergency work where the mortality may be easily gauged as over 10 per cent.

The mode of death in general peritonitis requires consideration. Vomiting and stagnation of intestinal contents are prominent symptoms, but they would hardly cause death without the toxæmia which reduces the volume of the pulse and gives rise to the ashen grey colour of the face. The contrast of a patient with severe vomiting but no toxæmia may be studied in a case of vicious circle, where the pulse remains good in spite of persistent vomiting. I have notes of such a case which occurred in 1906. Again, stagnation in the alimentary tract with distention is usually overcome if there is no general peritonitis, and in most cases it does not produce the depression of toxæmia.

### TECHNIQUE OF LYMPHATICOSTOMY.

A sand-bag or firm pad is placed under the upper dorsal spine, and the head drawn back and to the right. A 3-inch incision is made along the outer edge of the left sternomastoid in its lowest part: by retracting it inwards and the omohyoid outwards, and a careful blunt dissection, the junction of the internal jugular vein with the left subclavian vein is easily found and may be maintained in a perfectly dry condition. This dry condition is important for discovering that delicate structure, the thoracic duct. It is to be found entering the posterior surface of the subclavian vein either at or near its junction with the internal jugular vein: its size is no greater than a very small vein; its colour is yellow and it is nearly transparent. When found, its identity may be verified by putting a small pair of Spencer Wells on it close to its entry into the large vein. Under these conditions it fills with yellow transparent fluid in less than a minute. Its distal end is tied off and a fine glass cannula is passed into the slit-up duct, and this severed and attached to the skin by a stitch. Failing the use of a cannula, which I have not yet found small enough, the slit duct is stitched lightly to the edge of the wound.

The points to be remembered are: (1) A dry wound is necessary; (2) A light touch is needed in dissection, for as the duct is much more delicate than any other structure I have met with in general surgery; (3) The duct is nearly empty owing to depletion of the patient by starvation and vomiting.

Professor Langley has assured me that the way to find the thoracic duct in dogs is to give them a good meal of fats two hours before the operation. The directly opposite physiological conditions in a depleted patient make it necessary to have an assistant and a good light. In one case (*Case 3*) I believe I mistook the fine lymphatic trunk from the cervical glands for the main duct.

There are at least three points about this severance of the main lymphatic duct which require consideration: (1) The possibility of healing; (2) The



ultimate new path adopted by the lymph; (3) The presence of toxins in the lymph.

1. In the three cases where a complete severance of the duct is believed to have been made, the amount of lymph was not excessive, not more than 1 or 2 oz. a day. It diminished rapidly after the first few days, and the wound, which was left widely open at first, closed naturally in from two to three weeks without any fistula. Accidental lymphatic fistulæ have been cured by various means, Morgan, of Chicago,<sup>2</sup> adopting rectal feeding with success. This imitates the depletion found in patients suffering from peritonitis.

2. The ultimate new paths for the lymph are variously stated. Costain believes that the right lymphatic duct is the alternative path. Wendel<sup>3</sup> and V. C. Pennell (Cambridge School of Anatomy) state independently that an alternative lymph channel is to be found into the left azygos vein. Clinical evidence supports an alternative route: (a) *Case 1* is in good health with no lymphatic œdema; (b) Patients who have had severe operations on the posterior triangle, or gunshot wounds with scarring, do not develop signs of lymphatic block.

3. The presence of toxins in the lymph must be accepted if Costain's figures for the operation in dogs are credited. In future cases it is my intention to collect the lymph by means of a cannula and obtain if possible direct evidence of its toxicity. So far, cultures from the lymph in these cases have proved sterile.

## CASE REPORTS.

### *Case 1.*—Appendicitis with general peritonitis.<sup>4</sup>

An agricultural labourer, age 48, was admitted to Addenbrooke's Hospital on April 15, 1924, with a history that on April 13 he awoke at 2 a.m., with severe abdominal pain. In spite of that he got up and milked the cows. His bowels acted, but he became progressively worse, with pain and vomiting, on April 14, and was admitted to hospital on the afternoon of April 15. At this time there was considerable distention of the abdomen, restricted respiratory movements, especially in the lower abdomen, surface hyperæsthesia in the left iliac region, and deep tenderness across the whole lower abdomen. Per rectum there was tenderness and a tense feeling in the pre-rectal pouch. Pulse 88, regular; temperature subnormal; tongue dry. A large faecal vomit occurred just before I examined him. Unable to make any diagnosis other than 'acute abdomen', I opened in the middle line and inspected numerous coils of small intestine, all of which were acutely inflamed, distended, and deep scarlet in colour, with occasional slight smears of pus. The appendix was perforated; around it there were no local conditions other than those general to the middle and lower abdomen. The appendix was removed and the wound closed without drainage, the severe distention causing the usual difficulties in such cases of abdominal wall closure. Having decided that this was a case where recovery by the usual methods of after-treatment was almost out of the question, I proceeded to perform Costain's lymphaticostomy.

I exposed the junction of the internal jugular with the left subclavian vein, and identified the thoracic duct by its position and by seeing it fill with beads of brilliant clear yellow fluid resembling thin honey drained from the comb. The duct was ligatured off from the vein, slit up, and stitched to the skin; the wound was left open and lightly filled with gauze. A culture from this lymph was proved next day to be sterile.

On April 16 the patient was progressively more ill; he was semi-conscious at 10 a.m.; pulse irregular and feeble. There was only a small exudation from the wound in the neck. Abdomen still distended. At 11 p.m. he was still semi-conscious, could just be roused; chyle was discharging more freely from the neck. Respirations 28, pulse 128. The next day he was fully conscious, the pulse had improved, though the abdomen was still distended. Chyle was pouring freely from the neck, a dirty white colour.

April 19.—The distention was decreasing; good result at last after numerous enemata and six doses of pituitrin at intervals. The neck was still discharging freely. Pulse good.

April 23.—The distention had gone. There was discharge of pus from the abdominal wound and sudden diminution of chyle from the neck. By April 26 the neck was practically dry; the bowels acted daily. Since April 17 the temperature had never been above 98.8°.

May 10.—The patient was discharged from hospital with both wounds healed and normally convalescent.

*Comment.*—The paucity of lymph in the first twenty-four hours in this case is explained by the fact that this man was depleted of fluid by starvation and by a large faecal vomit; it was only after continuous rectal saline for twenty-four hours that lymph began to flow freely. No measures were taken to stop the flow of lymph, and this subsided in a week.

A year later the man is in normal health.

#### *Case 2.*—Puerperal septicaemia with general peritonitis.

A. W., age 24, admitted under Dr. J. Campbell Canney on third day of septic puerperium after confinement (midwife), April 27, 1924. Pain in lower abdomen, vomiting, temperature 103.4°. Immediate local uterine treatment.

Fourteenth day: temperature has varied from 102° to 99°; left knee effusion; abdomen distended. Fifteenth day: abdomen more distended, temperature 101°, pulse 120. Dr. Canney called me in with a view to lymphaticostomy. The abdomen was enormously distended and tender, with no liver dullness.

*OPERATION.*—The thoracic duct was exposed, ligated distally, and the proximal part incised and sutured to the wound. The abdomen was opened; a large quantity of free gas and some pus escaped. A condition of general peritonitis with thin pus; culture, *B. proteus* and streptococcus. Rubber drain left in. Pus and gas continued to discharge, and on the twenty-second day, when the wound was opened up again, a faecal fistula developed.

The lymphaticostomy hardly drained on the first day; more fluid discharged on the second day and for one week. The general condition appeared to improve somewhat after the lymphaticostomy and abdominal incision until the twenty-fifth day, when the respirations increased to 40 and 50, and the patient died on the thirty-second day with signs of pneumonia.

#### *Case 3.*—Appendicitis with general peritonitis in a girl.

J. D. A., age 4. Admitted Nov. 27, 1924, with a history of five days' illness, vomiting and abdominal pain.

On admission, pale, dusky complexion. Pulse ? 180, temperature 100.2°. Tongue coated with black fur (? bismuth). Abdomen very rigid, distended, tender all over, especially in right iliac fossa. Liver dullness diminished.

*OPERATION.*—Appendicectomy; appendix gangrenous. Generalized pus in abdomen. Tube drainage. Lymphaticostomy performed; the lymph duct severed was very small and never gave a satisfactory flow of lymph, although two or three draehms drained out daily for a few days. The child gradually improved and the temperature fell to normal in fourteen days. About the fifteenth day a secondary abscess discharged itself through the wound. The child left the hospital all healed on Jan. 8, 1925.

*Comment.*—The general severity of the case and high mortality in young children led me to perform what I believed at the time was a lymphaticostomy. The poor flow of lymph makes it more than likely that the lymph vessel from the cervical glands was opened.

*Case 4.*—Appendicitis with general peritonitis.

F. S., male, age 31. Onset with acute abdominal pain, April 16, 1925.

Admitted April 18, 1925. Patient collapsed, in great pain, with subnormal temperature. Board-like rigidity all over abdomen; pain referred chiefly to right iliac fossa.

*Operation.*—Right pararectal incision caused a large quantity of foul-smelling purulent fluid to escape; appendix completely gangrenous. All visible coils of intestine acutely inflamed with no closing off of purulent area. Tube drainage. Lymphaticostomy performed.

April 29.—There has been quite satisfactory drainage of lymph from the neck until to-day, when flow is only slight. Patient is now apparently making a good recovery, but temperature is  $100.2^{\circ}$  at night and at times there is distention of the abdomen. Per rectum a soft indefinite swelling.

May 2.—Patient feels very well and is able to read. Rather more swelling felt per rectum. 7.30 p.m.: sudden urgent dyspnoea and death from pulmonary embolism.

*Comment.*—This was a very severe case; toxæmia with board-like abdomen, unlikely to recover. The patient reached semi-convalescence, and death from pulmonary embolism on the fifteenth day after operation is no proof that the lymphaticostomy did not help him considerably. Possibly if the swelling in the pelvis, which was just palpable, had been drained, pulmonary embolism would not have occurred.

#### SUMMARY OF THE FOUR CASES.

1. Recovered, apparently as a result of lymphaticostomy.
2. Died of pneumonia and general exhaustion seventeen days after lymphaticostomy, which had produced some improvement.
3. Recovered in spite of lymphaticostomy only producing a slight flow of lymph.
4. Recovered from all acute symptoms, and when convalescent died on the fifteenth day of pulmonary embolism.

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## A CONSIDERATION OF THE HYPOPHYSIAL ADENOMATA.\*

BY NORMAN M. DOTT, EDINBURGH, and PERCIVAL BAILEY, BOSTON.

WITH A PREFATORY NOTE BY HARVEY CUSHING, BOSTON.

### PREFATORY NOTE.

It is highly profitable at times for a chief of service to play the part of bystander and to see what interpretations younger and fresher minds may put upon matters in which he perhaps has somewhat fixed ideas. In assembling for publication one's own familiar case histories, impressions and opinions may be read into them, albeit unconsciously. Those who peruse these histories for the first time, never having seen the majority of the patients, are at least spared from any such prejudices in their handling of the material.

On this basis, Mr. Dott and Dr. Bailey have made the accompanying review of the cases discharged from the Brigham Hospital Clinic with verified pituitary adenomata. They give some new interpretations of these lesions and arrive at some novel conclusions, particularly in their creation of a third or mixed type of adenoma with a corresponding clinical syndrome which shows a variable overlapping of the symptoms attributable on the one hand to hyperfunction and on the other to hypofunction.

Even without the tell-tale signs of tumour, there is no mistaking a definite case of acromegaly nor a definite example of pituitary insufficiency, but when we begin to split hairs over the intermediate clinical types which have been classified under the term dyspituitarism, we find ourselves in difficulties. We may see among these patients with pituitary adenomata overgrown individuals who are at the same time adipose and sexually dystrophic. On the other hand, as in one case the authors have chosen to report, we may see individuals who are skeletally undersized and yet show no genital dystrophy. Moreover, they may be either fat or lean. In short, we are dealing with a complicated matter affecting growth, metabolism, and sexual development in which there is undoubtedly some intimate interplay between hypophysis and gonads.

Since the publication of my monograph in 1912 there has been no general assembly of cases from the Clinic, and, as the authors say, it is probably wise at present to eliminate from discussion the many hypophysial disorders due to other than an actual glandular adenoma and to devote one's attention solely to the syndromes which accompany this particular lesion.

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\* From the Surgical Clinic of the Peter Bent Brigham Hospital, Boston, Massachusetts, U.S.A. Presented before the Surgical Section of the American Medical Association June 12, 1924.

It is somewhat disconcerting to realize how little advance in our knowledge of these disorders has been made during the past twelve years.

*On the experimental side* we have learned that extracts of the posterior lobe, contrary to the early interpretation of the physiologists, actually cause an oliguria rather than a polyuria, and this information has come to be applied in the treatment of diabetes insipidus. A somewhat academic discussion has arisen concerning the relation of diabetes insipidus to a lesion affecting the hypophysis versus one affecting certain adjacent nerve centres in the hypothalamus. But what is of greater importance, Evans has given the first experimental support to what has heretofore been merely an assumption—that the overgrowth which characterizes gigantism, and probably acromegaly as well, is, as long surmised, actually an expression of hyperpituitarism.

*On the clinical side* there has been a great improvement in cranial roentgenology, which has made it possible not only to have a better understanding of the deformities of the sella tureica, but also to detect, in many cases, the suprasellar tumours of congenital origin which arise from relics of Rathke's pouch and which in childhood produce the standard examples of so-called Frölich's syndrome. There has also been a great improvement in our operative procedures, a result of the surgeon's increased experience in exposing these lesions; but beyond these few things our progress in the twelve years has been slight. We are far from having any exact knowledge of the chemistry of pituitrin—still farther from any knowledge of the active principle (or principles) of the anterior lobe which influences growth. These objectives will be attained in due course, but there are many obstacles to surmount.

In the before-mentioned monograph, a parallel was drawn between these hypophysial disorders and corresponding thyroid disorders, and I do not know that we can do better to-day, for we are not entitled to base too much clinical importance on histological studies, even such precise ones as Dr. Bailey has made. It will be recalled that our understanding of exophthalmic goitre as a state of hyperthyroidism goes back to Halsted's studies of experimental thyroid hypertrophy, and that Halsted himself, after twenty years, was unable to repeat successfully his early experiments, and expressed himself as probably having misinterpreted them. As shown by others, his original interpretation, not his later one, was correct. Judgement is difficult and experiment fallacious.

From a therapeutic point of view, we at present stand in regard to hypophysial disorders about where Kocher and Reverdin stood when they began to operate on patients with goitre to relieve them from dyspnoea and other mechanical effects of the tumour. In the thirty years which have elapsed great strides have been made in our understanding of the disorders of thyroid function, and much of our knowledge has been acquired as the indirect outcome of surgical procedures. It is probable, however, that operations for thyroid disease of one sort or another have reached their peak. We have not only learned much regarding prevention, but even when goitre is established we now know that there are other methods of treatment which prove effective in many cases.

Though the problem is more difficult, it is not at all unlikely that the

surgery of the pituitary adenomata will run a similar course; and, profiting by the experiences of the past with thyroid disorders, we may hope it will be a shorter one. At present, however, about the best we can do is to combat by some operative measure the loss of vision brought about by pressure of the tumefaction upon the chiasm. We know nothing of the underlying causes, so that prevention is beyond our grasp, but we are at present encouraged to believe that certain of the adenomata are amenable to radiation. This, however, as seems to be true of the thyroid adenomata, may prove a disappointment, and that accidents may occur from too vigorous radiation is indicated by the experience in the case which the authors have selected in order to illustrate the condition of gigantism.

It is such a combination of clinical and pathological studies as Mr. Dott and Dr. Bailey have made that is most likely to accelerate not only advances in diagnosis and treatment, but—what is more important—a better understanding of the underlying factors provoking these diseased states.

HARVEY CUSHING.

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## I. INTRODUCTION.

In papers dealing with the subject of hypophysial lesions it has been customary to merge the many disorders of the pituitary gland in a general discussion of their symptomatology, pathology, and treatment. We do not presume to decry such discussions of past years. They have thrown much light upon a subject which was dark, and which still presents many obscure places, just as did discussions upon 'fever' as a disease entity in former times. However, it is our belief that the knowledge of pituitary disease has now reached a point at which it is advantageous to depart from this custom, and to select a specific lesion of the hypophysis for consideration of its various aspects. To this end we shall deal in this communication with the pituitary lesion of most frequent occurrence—the *hypophysial adenoma*.

It is idle to speculate upon who may have first described one of these adenomata, for in the earlier writings the terminology was so obscure that

many hypophysial tumours were regarded as 'sarcomata'. Indeed, much of the recent literature shows little advance in this respect. To Benda<sup>2</sup> great credit is due. He dispelled much of the obscurity in which neoplasms of the gland were enveloped, by his stress on the fact that histological methods which are adequate to display the normal structural characters of the gland must be employed in the study of its morbid states. In short, for the differentiation of these tumours histological methods must be employed more specific than the customary routine hematoxylin and eosin stains after fixation in Zenker's fluid. By the use of special methods Benda showed conclusively that many of the so-called 'sarcomata' of the hypophysis were composed of specific pituitary cells, and that therefore these tumours were adenomata.

The present communication is based upon the histological study of the 162 hypophysial adenomata in the Brigham Hospital series (to May 1, 1924) and upon a review of the clinical records of the patients from whom the specimens were obtained.\* The histological material was secured at operation in all but one instance, and we find ourselves in a position to differentiate certain types of these adenomatous tumours and to link with each of them a distinct clinical syndrome. The relationship between the histological findings and the constitutional aspects of the disorders in question has been practically constant in the entire series of cases.

## II. PATHOLOGICAL ANATOMY.

**Gross Appearances.**—The anterior hypophysis (pars distalis) is structurally akin to other glands of internal secretion, notably the thyroid and adrenal glands, and its adenomata present characters similar to corresponding tumours of these other organs. The hypophysial adenoma is a soft homogeneous mass of epithelial tissue, varying in colour from a white, brain-like appearance to a dark maroon colour. It is usually of a pale yellowish or pinkish tint. Like other adenomata, its rate of growth may outstrip its vascular resources, and it is often subject to local degenerative and hæmorrhagic changes on this account. Consequent upon these processes the formation of cystic cavities is not uncommon. Of the 162 cases in the present series, 28 contained cysts large enough to attract notice during operation.

While the hypophysial adenoma does not differ essentially in its general structure from the same tumour in other organs, its confined position has a striking influence on its behaviour and appearance. A thyroid adenoma as a sharply defined, circumscribed, globular mass can grow to large dimensions without encountering serious resistance. The hypophysial adenoma, on the other hand, encounters resistance from its very inception. With the intrinsic proliferative power of a neoplastic formation it begins to enlarge in its confined cell, bounded by resistant structures—bone and dura mater. The least resistant structure which the tumour meets is its parent gland, the hypophysis, which is largely or completely destroyed by compression. But ere long the

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\* The cases in Dr. Cushing's Johns Hopkins series, many of which comprised the basis of his monograph (1912)<sup>1</sup>, are not included in this study.

dural capsule becomes distended and bulges upward under the chiasm. Even the bony sella itself gives way to the persistent pressure; it becomes enlarged, ballooned out, and its walls undergo pressure absorption. This is the usual condition in the majority of the cases with the growth still confined within its distended dural and bony enlacement.

If left to itself, however, the tumour, still persistently enlarging, now takes the path of least resistance, which varies in individual cases. Commonly the limiting diaphragm of dura above it becomes attenuated and finally gives way, permitting the growth to enter the cranial cavity. This initial intracranial extension is often median, and takes place anterior to or sometimes behind the optic chiasm. In other cases it extends laterally, either free in the intradural space, or the growth may insinuate itself between

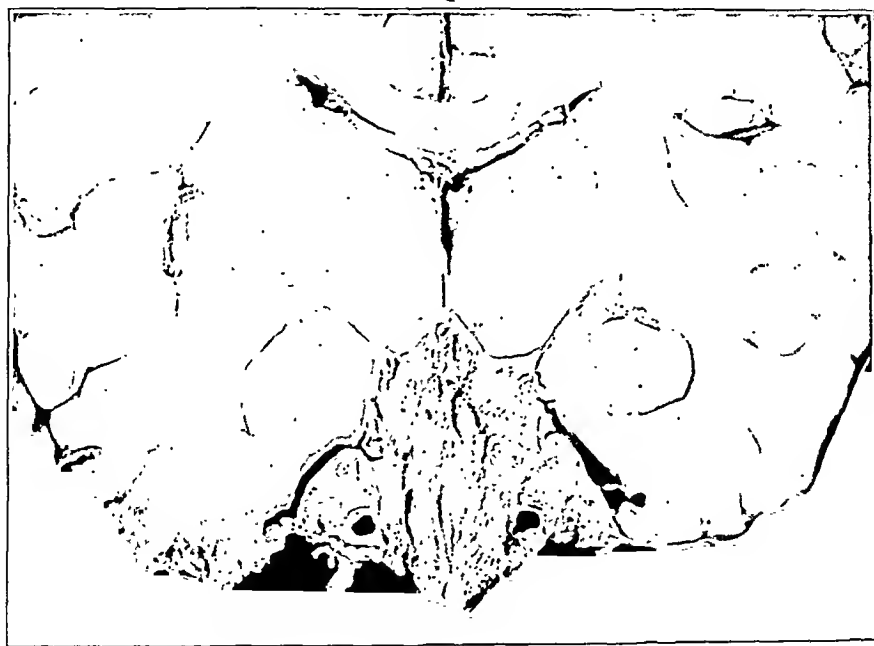


FIG. 222.—Coronal section of a brain, showing chromophobe adenoma. Note carotid arteries embedded in tumour.

the dura and the bone of the temporal fossa. On the other hand the upper diaphragm may remain intact, and the growth, through pressure erosion of the sellar floor, may project downward into the sphenoidal cells, where it is found covered by little more than shreds of the dural capsule, a few thin flakes of bone, and the mucous membrane of the cells.

The extensions that these simple adenomata will make under the influence of confinement are at times truly extraordinary, and have often given rise to the erroneous belief that the tumours are of a malignant nature. In one instance such an outgrowth extended between bone and dura across the temporal fossa, eroded the squamous portion of the temporal bone, and appeared outside the cranium beneath the temporal muscle. Not uncommonly in its



lateral extension the growth embeds and surrounds the extradural portion of the internal carotid arteries (*Fig. 222*), and may even extend into Meckel's cave.

Its intradural extensions in advanced cases are no less remarkable. It usually projects upwards as a rounded mass, elevating the optic chiasm, while the optic nerves and tracts are deflected on to its lateral surfaces, and, stretching round it, are compressed and flattened. It sinks its summit deeply into the base of the brain, sometimes compressing the hypothalamic region to the extent of obliterating the third ventricle, at other times indenting the uncinate region on either side. Under the influence of increasing compression and confinement, processes of the tumour may extend into every available crevice: upward, between the brain-stem and temporal lobe; backward, as a thin tongue-like process over the surface of the tentorium or through the hiatus tentorii to the cerebello-pontine angle.

Though these simple adenomata, as will be seen, show unmistakable differences in their finer microscopic structure, to the naked eye they are indistinguishable.

**Microscopic Structure.**—The pars distalis of the normal hypophysis, it will be recalled, is composed of columns of polygonal cells between which are found small blood-channels supported by a scanty connective-tissue framework. The cells may be divided into two main classes in accordance with their differing affinities for dyes. We thus distinguish the chromophil cells, which have such an affinity, from the chromophobe cells, which do not. The chromophil cells are subdivided by the specific staining affinities shown by the granules contained in their cytoplasm. One type of granules stains with cosin, acid fuchsin, neutral dyes, etc.; they are usually known as the eosinophilic granules. The other type stains with alum hematoxylin, kresofuchsin, acid violet, etc.; and they are commonly designated basophilic granules. The terms acidophilic, versus basophilic, are relics of a time when one type of granules was erroneously supposed to have an affinity for acid dyes and the other for basic dyes. It is preferable, therefore, to use some non-committal designation, and we shall refer to them as *alpha* or *beta* granules (*Fig. 223, A*).

Many synonyms exist for these cell types—(1) *Chromophobe* = chief cells, reserve cells, siderophil, etc.; (2) *Chromophil*: (a) *Eosinophil* = acidophil, fuchsinophil, oxyphil, etc.; (b) *Basophil* = cyanophil, etc.—in accordance with theories of their staining reactions or with varying views regarding their function. From each of them adenomata may take origin. The chromophobe adenoma is by far the most common; the eosinophilic tumour is not infrequent; the basophilic is rare, and is known to exist only as a minute, almost microscopic nodule.

Loewenstein<sup>3</sup>, who has made a careful study of the site of origin of hypophysial adenomata, states that they are encountered most frequently in one of three situations in early stages of their development: (1) In the pars intermedia; (2) In the peripheral part of the pars distalis; and (3) In the vicinity of the stalk (pars tuberalis). It is in these parts that the chromophobe cells predominate; and the fact that they are embryologically the least highly differentiated cells of the hypophysis perhaps explains both the

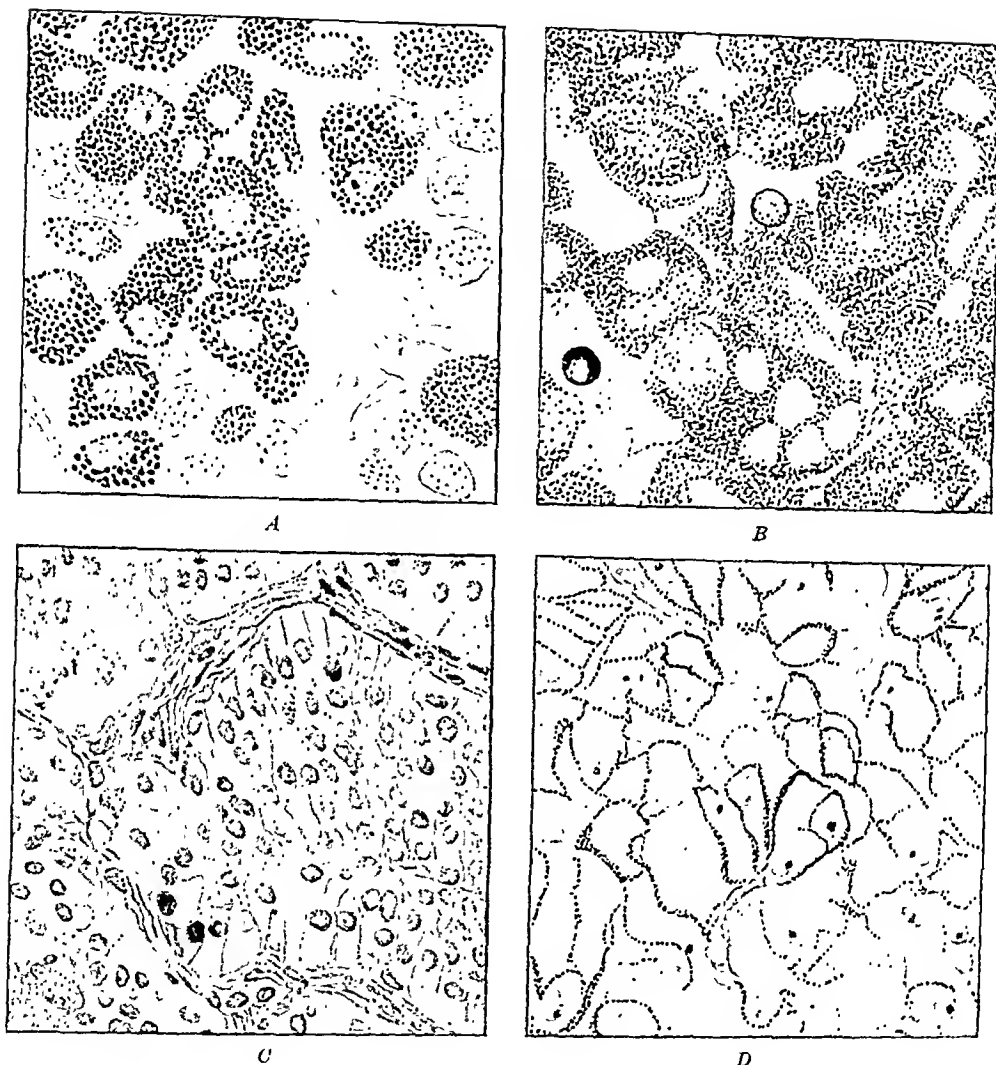


FIG. 223.—A. Normal pars distalis. Acid fuchsin-acid violet stain on Regaud-fixed tissue. Alpha granules red; beta granules dark blue; other structures pale blue.

B. Typical adenoma from a case of acromegaly. (P.B.B.H., *Surg.* No. 15505.) Neutral ethyl-violet-orange G stain on Regaud-fixed tissue. Alpha granules violet; other structures orange.

C. Typical chromophobo adenoma from a patient with 'hypopituitary' syndrome. (P.B.B.H., *Surg.* No. 19512.) Hematoxylin and eosin stain on Regaud-fixed tissue. Such an adenoma stained as A, B or D would show no granules whatsoever.

D. Adenoma from a patient with a mixed clinical syndrome. (P.B.B.H., *Surg.* No. 19907.) Neutral ethyl-violet-orange G stain on Regaud-fixed material. Alpha granules violet; nuclei slightly stained; other structures orange.

All drawn under oil immersion lens except C, which is magnified 300 diameters.

frequency with which adenomata originate in these places and the predominance of the chromophobe type of tumour.

**The Chromophobe Adenoma.**—This variety of adenoma is associated with evidences of glandular insufficiency. It is by far the commonest tumour, the series containing 107 examples. On a structural basis two types are perhaps deserving of mention: (1) Those in which the columnar structure of the normal hypophysis is maintained in greater or lesser degree; and (2) Those consisting of a mass of cells without architectural arrangement and containing very little connective-tissue stroma. These latter adenomata have sometimes been called 'strumas'.

In the chromophobe elements of the normal hypophysis, areas may be seen where the cell boundaries are indistinct—the so-called 'Kernhaufen'. It is possible that the adenomata of the second type arise from these areas, but the difference in structure is probably to be accounted for by the varying proliferative activity of individual tumours. The adenomata grow by multiplication of cells of the hypophysial columns, which therefore enlarge. These enlarged cell columns are then invaded by capillaries which provide for their adequate nutrition, and in some cases they thereby become divided off into well-formed secondary columns. Those cells of the chromophobe tumours which rest on connective-tissue septa are usually of an elongated, cylindrical epithelial type (*Fig. 223, C*), but elsewhere they are apt to be spindle-shaped or polygonal in form. In some of the soft tumours, on the other hand, which have no connective-tissue septa, the cells may appear circular and almost lymphoid in character.

Clinically, this form of chromophobe tumour is the type that is associated with the 'hypopituitary' syndrome. In the tissues from no case which was clinically free from recognizable acromegalic characteristics have we been able to demonstrate any cells containing  $\alpha$  or  $\beta$  granules, with the occasional exception of a rare, isolated cell brought into view after a search through many sections.

**The Chromophil Adenoma.**—*The Eosinophilic Tumour.*—This adenoma is the form which accompanies the hyperpituitary syndrome. Material from 39 cases is available for study. The tumour in some instances is composed wholly of eosinophilic cells (*Fig. 223, B*), no cells free from  $\alpha$  granules being demonstrable when proper staining methods are used. In the majority of cases, however, though the adenoma be not *purely* eosinophilic, cells containing eosinophilic granules greatly predominate. But even under these circumstances the non-granular cells do not present appearances identical with the cells of the chromophobe adenoma; they are apt to be larger, to have an abundant coarse cytoplasm, and multinucleated cell forms of either type (granular or non-granular) are frequently seen. As a rule these adenomata are of the 'strumous' variety with scant connective tissue, and they are prone to cystic degeneration. In our experience tumours of this type are constantly associated with the syndrome of overgrowth, i.e., acromegaly or gigantism.

*The Basophilic Tumour.*—This tumour hardly finds a place in a clinical paper, and we mention it here merely for the sake of completeness. As stated above, it occurs only in the form of minute intraglandular nodules, and, so

far as we know, gives rise to no clinical manifestations. There are no examples in the series.

**The Mixed Type of Adenoma.**—In 13 cases in the series an adenoma of a mixed type has been encountered. Three varieties may be distinguished: (1) The cells resemble those of the chromophil (eosinophilic) adenoma, but either contain no  $\alpha$  granules or a few scattered granules only are discernible; (2) The structure resembles that of the chromophobe adenoma, but each cell shows a ring of  $\alpha$  granules in its periphery (*Fig. 223, D*); or (3) The chromophobe adenoma type again predominates, but the cells contain granules which stain slightly by the eosinophilic dye. These tumours prove to be associated with syndromes in which clinical evidences of both hyper- and hypopituitarism are present. In other words, a mixed clinical type appears to exist in which the features of these two well-recognized conditions are blended in varying proportions and in which the structure of the adenoma reflects the character of the clinical picture.

**The Malignant Adenoma.**—Hypophysial adenocarcinomata are rare, and the present series contains only 3 examples. In these three cases the constituent cells have shown no granules. Possibly the chromophobe cells, being less highly specialized forms, are more prone to malignant transformation than are the eosinophilic or basophilic elements.

The tumours are varied in structure. At one part distinct alveolar formations are found, closely resembling those of the thyroid gland, but without basement membrane, and sometimes two or more cell rows in depth. In other parts scattered or closely packed masses of cells occupy irregular spaces in the connective-tissue stroma. Everywhere the variety in size and shape of the cells is striking, and all grades of degenerative change are widespread. Mitoses are frequent. Tumour cells, in irregular colonies, may be observed wedging themselves between strands of fibrous tissue and actively invading it.

These malignant adenomata, like other malignant tumours, possess metastasizing as well as invasive qualities. In the only one of our three cases in which post-mortem information on the point was obtainable, metastases were present in the liver; the hepatic lymph glands were also involved and were probably secondarily invaded from the liver. The metastatic growth presented a structure identical with that of the original hypophysial tumour.

### III. THE CLINICAL SYNDROMES.

Apart from the neighbourhood signs and symptoms produced by pressure of the tumour on adjacent anatomical structures, which may result from any hypophysial adenoma regardless of its histological structure, there occur certain striking constitutional changes in the individual. These chiefly affect growth, metabolism, and the deposition of fat, together with changes in other and allied glands, notably in the gonads which lead to alterations in the primary and secondary characters of sex.

These constitutional manifestations of the adenomata are modified by many factors: by the type of the adenoma; by the degree in which the normal glandular function is interfered with; and by the relation of the onset of the

disorder to age. Admittedly, there are some patients even with large adenomata in whom these secondary derangements might pass unnoticed were it not for the existing tumour; and other cases in which apparently identical bodily changes occur without clinically demonstrable tumour. Nevertheless, it is far more common for the hypophysial adenomata to be accompanied by constitutional syndromes sufficiently characteristic to justify their separation into clinical groups. In describing them we shall employ the designations coined by Cushing in 1909,<sup>4</sup> viz., hyperpituitarism, hypopituitarism, and dyspituitarism. By the selection of recent examples which have come under our personal observation in the Clinic, we shall endeavour to illustrate the several types in accordance with the following classification:—

*A. Tumour symptoms with slight constitutional disturbance.*

*B. Outspoken 'hypopituitary' syndrome:* (1) Infantile type; (2) Adult adiposo-genital type; (3) Wrinkled atrophic type.

*C. Outspoken 'hyperpituitary' syndrome:* (1) Gigantism; (2) Acromegaly.

*D. Mixed or 'dyspituitary' syndrome.*

This terminology is not entirely satisfactory. We do not believe that all the symptoms shown by the patients we have grouped under 'hypopituitary' syndromes are due to suppression of the function of the hypophysis. Moreover, although gigantism—which Krumpholtz terms hyperprepituitarism—is apparently a manifestation of increased activity of the pars distalis, acromegaly is not so certainly due to the same cause, but may, as often stated, be the result of a perverted secretion.

Furthermore, being doubtful whether adiposity is actually due to an hypophysial lesion, and wishing to include the group of non-obese cases to which Cushing for some years has called attention in his clinics, we were unable to use the term dystrophia adiposo-genitalis as synonymous with loss of hypophysial function. We were tempted, therefore, to introduce a new term, 'raripilogenital syndrome' (*L. raripilus*, scanty hair), which would cover both the adiposo-genital and wrinkled atrophic types, emphasizing the symptoms common to both—namely, the cutaneous and genital changes—but we repented in time of our desire further to complicate a subject already overburdened by terminology.

In the main we believe the concepts of hypo- and hyperpituitarism are correct, and with the reservations we have just made we shall continue to use them. The development of these concepts is interesting. They of course had their beginning in 1886, when Marie<sup>5</sup> established the clinical entity which he called acromegaly, and when Minkowski<sup>6</sup> in the following year pointed out the relation of the disorder to the hypophysis. Though Marie first regarded the syndrome as due to hypofunction of the hypophysis, Massalongo<sup>7</sup> considered it to be due to a continuance in post-fœtal life of the function of the hypophysis and thymus. In the same year he pointed out the relationship of gigantism to acromegaly, a view which was further developed in 1895 by Brissaud and Meige.<sup>8</sup> Meanwhile efforts were made by Horsley and others to reproduce these states or their counterpart in the laboratory by pituitary extirpations. These efforts were ineffectual, and naturally enough the idea as expressed by Tamburini<sup>9</sup> in 1894, that acromegaly, at least in its early phase, was an expression of glandular hyperactivity, began to prevail.

During this time pituitary tumours accompanying acromegaly had unfortunately been described under a variety of terms, as sarcomas, gliomas, cylindromas, etc., and it is to Benda's credit that in 1900 he made clear that they were all adenomata of the gland. A year later, from a study of the hypophyses from four cases, he, with Fränkel and Stadelman,<sup>10</sup> came to the conclusion that the hyperplasia of the chromophil cells which was present in all of them probably indicated an increased activity of the gland. The interpretation of this finding as an evidence of hyperfunction was without experimental control—such as Halsted's experimentally produced hypertrophy in the case of the thyroid—and of course was purely a matter of conjecture.

During the next several years confusion was added to the subject by the report of certain cases of pituitary tumours, many of them adenomata, in which no recognizable symptoms of acromegaly occurred. One of the first of these was reported in 1900 by Babinski.<sup>11</sup> He ascribed to a lesion of the hypophysis the genital hypoplasia which he had observed in a young girl; but he does not seem to have grasped the idea that the condition might be due to hypofunction. He noted that his patient was somewhat obese, but did not call especial attention to this fact. Fröhlich<sup>12</sup> a year later, in a report of a single case, emphasized the adiposity, and, though he recorded some hypoplasia of the genitalia, in summarizing his syndrome he mentioned specifically only the adiposity and the cutaneous changes together with the tumour—the presence of which was betrayed by the defect in the visual fields—as the essential features of the condition. Nowhere does he suggest that these symptoms might be due to hypofunction of the hypophysis.

Bartels,<sup>13</sup> in 1906, seems to have been the first to couple adiposity and genital hypoplasia as the essential features of the clinical syndrome which he called 'dystrophia adiposo-genitalis', but stated that a lesion of the hypophysis stood in no causal relationship to the syndrome. About the same time ophthalmologists, gynæcologists, and neurologists (Axenfeld, Miller, Cushing, Kon, and others) had begun to report cases in which adiposity, amenorrhœa, or sexual dystrophy were associated with pituitary tumours.

An explanation of these various and confusing syndromes had to await the experimental reproduction of allied states in the lower animals. It was not even known at this time that the hypophysis was anything more than a vestigial organ as unimportant to the economy as the pineal body or thymus. This view was generally held until 1907, when Paulesco<sup>14</sup> reported a series of operations on the gland by a new method of approach which showed that its complete removal was incompatible with the maintenance of health or perhaps even of life. These experiments were repeated in the following year by Cushing<sup>15</sup> and his collaborators, and they were the first to point out (1909) that a condition resembling adiposo-genital dystrophy was a consequence of partial hypophysectomies in the adult dog. A year later Aschner<sup>16</sup> showed that skeletal infantilism could be produced by similar operations on puppies. These observations for the first time furnished a dependable proof that lowered function of the hypophysis was the primary factor in the various and imperfectly understood clinical syndromes which had been previously ascribed.

Though by the injection of its extracts physiologists had shown the presence of an active principle in the posterior lobe, its removal was unattended by obvious consequences. On the other hand, as noted above, it had long been recognized that definite symptoms followed experimental injury to the anterior lobe—dwarfism, genital hypoplasia, etc., but the negative results of injections of its extracts failed to confirm the association between the anterior lobe and these symptoms. Nevertheless, it was assumed that these symptoms both in the laboratory and clinic were due to hypophyseal insufficiency, while as a natural corollary gigantism and acromegaly were associated with glandular over-activity. Though this had been previously conjectured, experimental proof was lacking until the recent studies of Evans and his collaborators<sup>17</sup> demonstrated that actual gigantism may be induced, and the dwarfism, etc., of experimental hypopituitarism relieved, in rats by the intraperitoneal injection of freshly prepared anterior-lobe extracts.

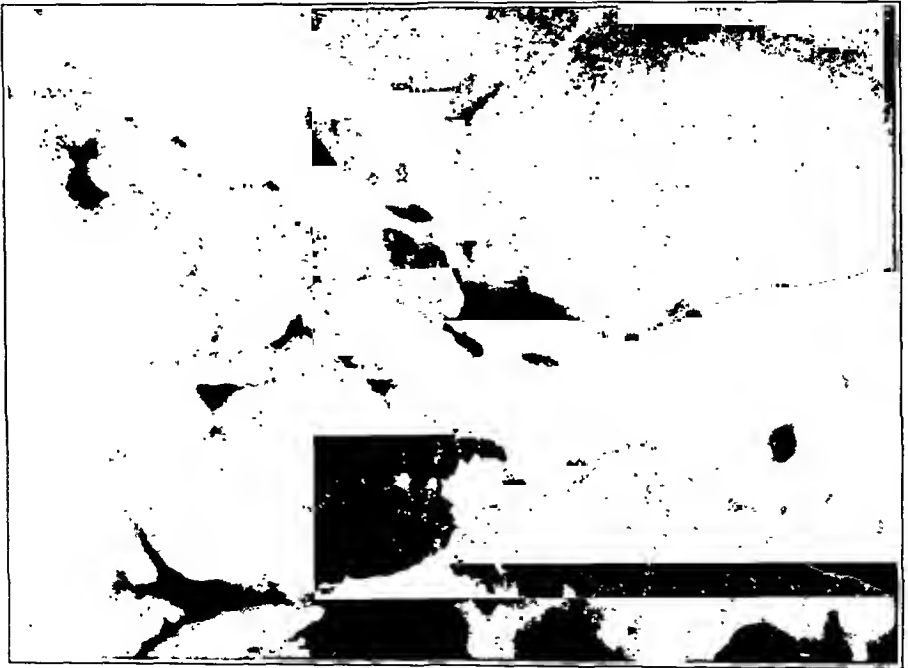


FIG. 224.—Case 1. To show the enlarged sella. ( $\times 1$ .)

## CASE REPORTS.

### 1. CHROMOPHOBE ADENOMATA.

**Chromophobe Adenoma with Slight Constitutional Disturbances.**—In presenting our illustrative cases, we may well begin with a chromophobe adenoma in its simplest clinical form—namely, a growth large enough to cause well-defined neighbourhood symptoms without provoking any but the most inconspicuous constitutional evidences of disturbed glandular function.

Inasmuch as the patient was a woman who had passed the menopause, there is missing in the clinical picture even the customary sign of symptomatic amenorrhœa.

*Case 1.*—*Surg. No. 20950.* Chromophobe adenoma in adult with local symptoms alone. Transphenoidal operation. Considerable improvement in vision.

**ADMISSION.**—March 18, 1924. Mrs. F. H., age 49, a housewife of English birth, referred by Dr. W. E. Bruner, of Cleveland, Ohio.

**CLINICAL HISTORY.**—She has lived an active, normal life with no significant illnesses. Married at 21 years, she has had four normal pregnancies—three healthy children alive and well, and one stillbirth. Her youngest child is 17 years old—i.e., her last pregnancy occurred in her 31st year. Her husband is living and well. She had no wish to avoid a further pregnancy, but none took place. Menstruation began at the age of 14 years and had been quite normal and regular until the age of 47 years (18 months ago), when it abruptly ceased—apparently quite a normal menopause. One year ago vision began to fail and has progressively deteriorated. She has herself observed that the defect involved the temporal fields of vision. Four months ago she began to see double, and her friends remarked that her left eyelid drooped slightly. These conditions persist to the present time.



FIG. 225.—*Case 1.* Photographs of patient.

She has always been stout, and during the past twenty years her weight has probably increased slightly, but not more than advancing years would reasonably account for. She has experienced no unusual thirst, nor has she noted any unusual frequency of urination. She has had no headaches.

**EXAMINATION.**—The following positive neighbourhood findings were revealed: a bilateral primary optic atrophy of moderate degree; V.O.S. 20/30; V.O.D. 30/40; bitemporal hemianopsia—the temporal defect not quite complete on the left side; history of diplopia, but no obvious strabismus. X rays show the sella turcica widely expanded (*Fig. 224*); the floor is irregular in outline and depressed into the sphenoidal sinus; the anterior and posterior clinoid processes are eroded.

She is a stout, vigorous woman of middle age (*Fig. 225*), intelligent and co-operative. Height 159 cm., weight 86.2 kilo. The skin is of normal texture, humidity, and colour. The hair is entirely normal in quality, quantity, and distribution. The basal metabolic rate is — 9 per cent. There is a slight, moderate, diffuse enlargement



of the thyroid gland, of frequent occurrence in the Lake district, where she lives. She presents no symptoms suggesting thyroid dysfunction. One X-ray treatment was given while she was awaiting her operation, with no subsequent appreciable change in the visual fields.

**OPERATION.**—March 28, 1924. Dr. Cushing exposed the bulging sellar floor by the usual sublabial and transphenoidal procedure which he has described. A sellar decompression was performed, and on incising the tense dural capsule of the tumour soft adenomatous tissue was extruded, and a considerable amount of this tissue was removed with a spoon from within the capsule.

**Post-operative Course.**—She made a rapid and satisfactory recovery from the anæsthetic. Subjective improvement in vision was noted within a few hours. Convalescence was uneventful. At the time of her discharge on April 11 her visual acuity was practically normal. A clear-cut right temporal hemianopsia remained. A second X-ray treatment was given twelve days after operation, and arrangements were made for six further treatments to be given at intervals of three weeks.

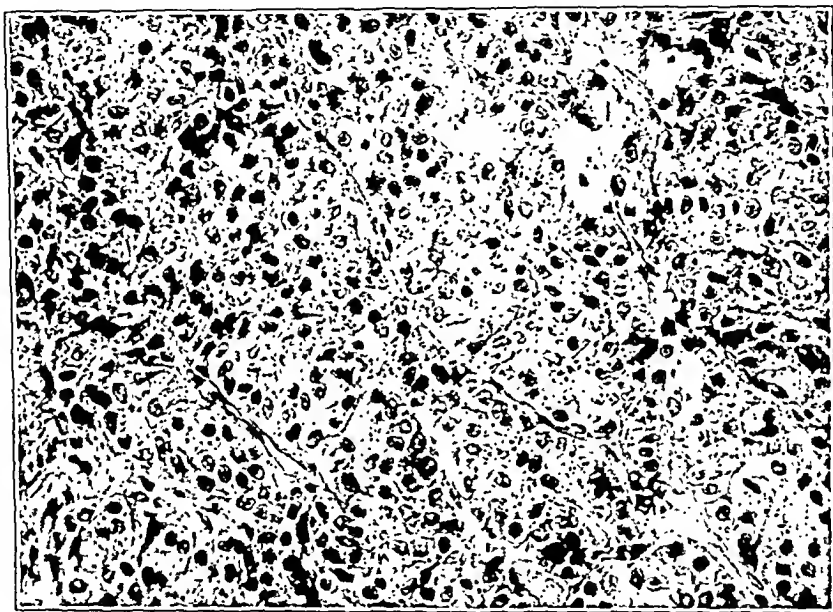


FIG. 226.—Case 1. Photomicrograph of chromophobe adenoma. (Hæmatoxylin and eosin stain.) ( $\times 300$ .)

A report of May 9, 1924, from Dr. Bruner, states that "the defect in the temporal field in the left eye is entirely gone, and in the right—which had almost a complete temporal hemianopsia—there is now only slight peripheral contraction—certainly a most gratifying change".

**PATHOLOGY.**—Sections of the tissue show the tumour to be composed of epithelial cells, arranged in columns from 2 to 6 cells in thickness, very much after the manner of the normal pars distalis (Fig. 226). These columns are separated by capillaries with thin walls. The cells have oval nuclei, and in their cytoplasm no granules are to be found except mitochondria.

**DIAGNOSIS.**—Chromophobe adenoma.

This, then, is an example of a chromophobe adenoma with unmistakable signs of its presence but with practically no constitutional indications of

hypopituitarism. It is true that she was somewhat obese, but this condition had been present for many years without particular change at the onset of her disease. It may be objected that the amenorrhœa should be interpreted as a consequence of the adenoma. In any other disease, however, the menopause occurring at the age of 47 would not excite comment. It is our impression that the pathological amenorrhœa of pituitary insufficiency is of gradual onset, and the abrupt cessation of the menses in this patient leads us to believe it was unconnected with the hypophyseal disorder which shortly followed it.

It is evident from the records that a large number of pituitary adenomata in women become manifest either shortly after pregnancy or the menopause; and we incline to the view that the physiological disturbance which these events are known to induce may act as a predisposing factor to adenomatous development in the hypophysis.

**Chromophobe Adenoma with 'Hypopituitary' Syndrome.**—This, as already stated, is by far the commonest type of pituitary disorder. Of the 162 cases, 107 fall definitely into this category. In each instance a chromophobe tumour was identified. The disease is essentially one of adult life. In the series only 3 cases were recorded in the second decade, the youngest being 14 years old. The numbers were fairly evenly distributed in the third, fourth, and fifth decades of life, and the great majority were credited to them. A considerable number, however, occurred with diminishing frequency beyond the age of 50 years. These figures represent the age of the patient on admission to the hospital; as the average duration of symptoms prior to admission was between three and four years, an adequate idea of the usual age at the onset of the malady may be gained. The sexes are apparently equally afflicted, there being 54 males and 53 females in the series.

The local manifestations of the tumour are the same for all ages, but its secondary or hypopituitary effects are modified considerably by the age of the individual. We give three examples of the disorder: as it appears in the young adolescent, in the young adult, and in the middle-aged patient. As stated, the disease is rare in the extremes of life, and the following case, which has come under our personal observation, is one of the three below 20 years of age. Though the secondary sex characters were well established, the glandular insufficiency occurred early enough to check skeletal growth.

*Case 2.*—*Surg. No. 18904.* Chromophobe adenoma of adolescence with partial infantilism. Mistaken for cyst of Rathke's pouch. Transfrontal operation. Considerable improvement in vision.

**ADMISSION.**—May 26, 1923. E. H. D., male, age 18, a student, referred by Dr. J. G. Janney, Dodge City, Kansas, with the complaint of failing vision and headaches.

**CLINICAL HISTORY.**—Though always a very quiet boy and disinclined for rough sports, he had developed quite normally, both physically and mentally, until the age of 15 years, when he practically ceased to grow, and his height has remained at about five feet. At the same time (that is, three years ago) he began to suffer from severe, 'bursting' headaches, which have increased progressively in severity and frequency. They have recently been accompanied by vomiting and followed by a marked degree of somnolence, lasting on occasions for twenty-four to thirty-six hours. Three years ago also, his vision began to fail; it has become gradually worse, and he has been aware of bitemporal blindness.

As a child, he had a good soprano voice, but at 15 it 'broke'; it, however, did not attain the normal male bass, but remains irregular and high in pitch. A year later, after what appears to have been a normal adolescence, his youthful libido was lost, and he observed that nocturnal emissions no longer occurred. He began to experience increasing difficulty in concentrating on his studies. During the past eighteen months he has noted increased thirst, has had to urinate more frequently during the day, and has had to rise at least once during the night on this account. Until a year ago he had been a very slim boy, and for some time previously his weight had remained about 38 kilo. During the past year he has gained 11.3 kilo. in weight. He has had occasional slight epistaxis, and his eyes have become more prominent in the past year.



FIG. 227.—Case 2. To show the ballooned sella. ( $\times 1$ .)

**EXAMINATION.**—The following positive findings were revealed: *Local.*—A bilateral primary optic atrophy, with bitemporal hemianopsia; V.O.S. 20/100; V.O.D. 20/50. The X-ray disclosed a widely distended sella its floor slightly depressed, and the posterior clinoid processes and dorsum sellæ extensively eroded (Fig. 227). Moderate bilateral exophthalmos.

*General.*—Owing to his short stature, slight skeletal frame, and moderate adiposity, he presents the appearance of a boy of fourteen (Fig. 228). Height slightly over 5 ft. (156.2 cm.); weight 49 kilo. He is quite beardless. The hair of the scalp, though abundant, is very soft, silky, and fine. The axillary hair is very scanty; on the pubes it is sparse and has a distribution of feminine type: the trunk and limbs are hairless. The skin is thin, dry, and pale, and of a fine, soft texture. He is not especially obese, but one notes the characteristic fullness of the breasts, hips, and

lower abdomen. The genitalia are normally developed. A slight degree of genu valgum is present. The basal metabolic rate is  $-8$  per cent. Observation in hospital shows that his fluid intake and his urinary output are slightly in excess of normal limits—up to 2600 c.c. daily.

A slight opacity just above the greatly enlarged sella could be made out on stereoscopic films, and a presumptive diagnosis was made of an intrasellar tumour of craniopharyngeal pouch origin—a more likely diagnosis than adenoma considering the youth of the patient. Consequently, Dr. Cushing decided to operate by the transfrontal route.

**OPERATION.**—June 9, 1923. A right osteoplastic flap was reflected, the dura raised from the orbital roof, incised along the sphenoidal ridge, and the frontal lobe, protected by its dura, was elevated to expose the optic nerves and chiasm. A reddish mass elevating the chiasm and projecting upwards between the optic nerves was disclosed, obviously an adenoma. The dural capsule was incised and a sufficient portion of the tumour was removed to free the chiasm and optic nerves from pressure. The bone flap was replaced and the wound closed in layers as usual.

**Post-operative Course.**—Recovery from the operation was uneventful, though there was a temporary increase in the polyuria, which amounted to 5000 c.c. on the fourth day, but gradually subsided to the pre-operative level. Vision improved remarkably, the acuity rising to V.O.S. 20/50, V.O.D. 20/20, and the fields expanding to normal limits within three weeks. Headache, vomiting, and somnolence were entirely relieved. One X-ray treatment was given fourteen days after the operation.

Reports from the patient and his physician subsequent to his discharge relate that he remained well and at work as an office boy until some five months later, when he again began to suffer from headaches, and at the same time his polyuria increased once more to between 3000 and 4000 c.c. daily. He was given two X-ray treatments with prompt and complete relief.

**PATHOLOGY.**—The tissues removed show (Fig. 229) the tumour to be composed of elongated epithelial cells, divided into definite cell columns by blood sinuses. The connective tissue is confined to the thin walls of these sinuses. The epithelial cells form in places a pseudostratified epithelium with the bases of the cells on the blood sinuses. The cytoplasm of the cells is vacuolated extensively, and no granules are to be found except mitochondria.

**DIAGNOSIS.**—Chromophobe adenoma.

This is the nearest approach to a case of infantilism in the series of hypophysial adenomata.\* Infantilism, when occurring in association with a hypophysial tumour, is so constantly related to a congenital tumour (tumour of Rathke's pouch) that in this case, although positive evidence of

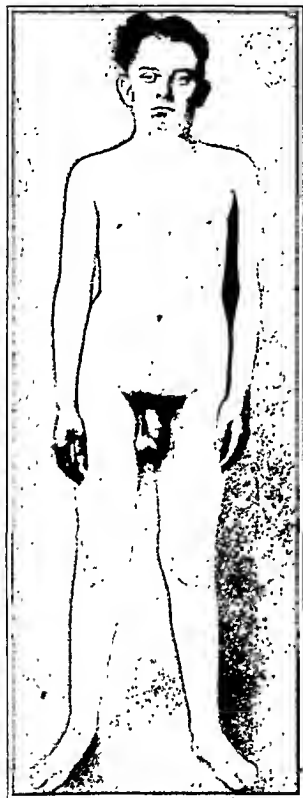


FIG. 228.—Case 2. Photograph of patient, age 18; height 5 ft.

\* Since this review of the clinical histories by Mr. Dott, there has been in the Clinic an example of typical chromophobe adenoma with ballooned sella in a child of 10 years with skeletal infantilism and adiposity, much more commonly associated with craniopharyngeal pouch tumours.—P. B.

a suprasellar lesion was lacking, a transfrontal operation was performed, whereas the operation of choice for hypophyseal adenomata with large ballooned sellae is by the transphenoidal route from below. The significance of polyuria will be discussed later; it is to be regarded as incidental, and not as a peculiar characteristic of this type of pituitary disease.

The following is a typical example of the disease in a young adult in whom there were constitutional evidences of glandular insufficiency of the adiposo-genital type. The case differs in no essential respects from some hundred others which fall in the central age group.

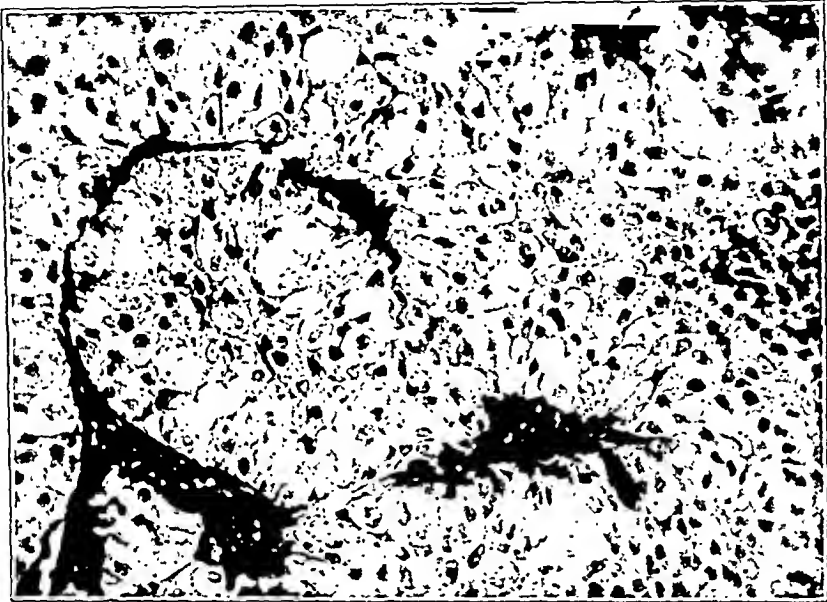


FIG. 229.—Case 2. Photomicrograph of chromophobe adenoma. (Hematoxylin and eosin stain.) ( $\times 300$ .)

**Case 3.**—Surg. No. 20323. Chromophobe adenoma with hypopituitarism in a young adult. Operation, with improvement in vision.

**ADMISSION.**—Dec. 14, 1923. Benjamin F., age 27 years, unmarried, a Jewish house painter, referred by Dr. J. R. Shannon, of New York City, with the complaint of failing vision and of headaches.

**CLINICAL HISTORY.**—He has always been rather obese, even as a boy; his parents and brothers and sisters are also rather stout—one sister remarkably so. During the past year he has not gained, but has probably lost slightly in weight. Fourteen months ago he awoke one morning to find that his vision had suddenly failed to an alarming degree. He was unable to read and noted that his lateral fields of vision were gone. Not long thereafter he observed that sexual libido, which had never been very active, was in total abeyance, and that his normal nocturnal emissions had ceased. Ten months ago he began to suffer from headaches, at first mild and generalized, but progressively increasing in severity, and recently referred to the back of the eyes. He has complained of loss of interest and ambition in his work, and of becoming easily tired.



FIG. 230.—Case 3. To show outline of sella. ( $\times 1$ .)



FIG. 231.—Case 3. Photographs of patient of adipose type.

**EXAMINATION.**—The following objective findings were revealed: *Local.*—Bilateral primary optic atrophy; V.O.S. 20/50; V.O.D. light perception only; bitemporal hemianopsia. X rays show the sella turcica greatly expanded, its floor depressed into the sphenoidal sinus, and its anterior and posterior clinoid processes eroded (*Fig. 230*).

*General.*—There is a moderate degree of somnolence; he is somewhat dull, heavy, stupid, and grossly obese (*Fig. 231*). There are heavy deposits of subcutaneous fat over the shoulders, lower part of abdomen, and hips. Height 165 cm., weight 111 kilo. The skin is fine and soft in texture. The hair of the scalp is sparse, fine, and silky. The beard is very scanty, as is the hair of the axillæ. The pubic hair has a definite, sharp, upper limit of feminine type. The genitalia appear normal. The temperature tends to be slightly subnormal. The pulse is slow, varying between 60 and 70 per minute. The basal metabolic rate is  $-8$  per cent.

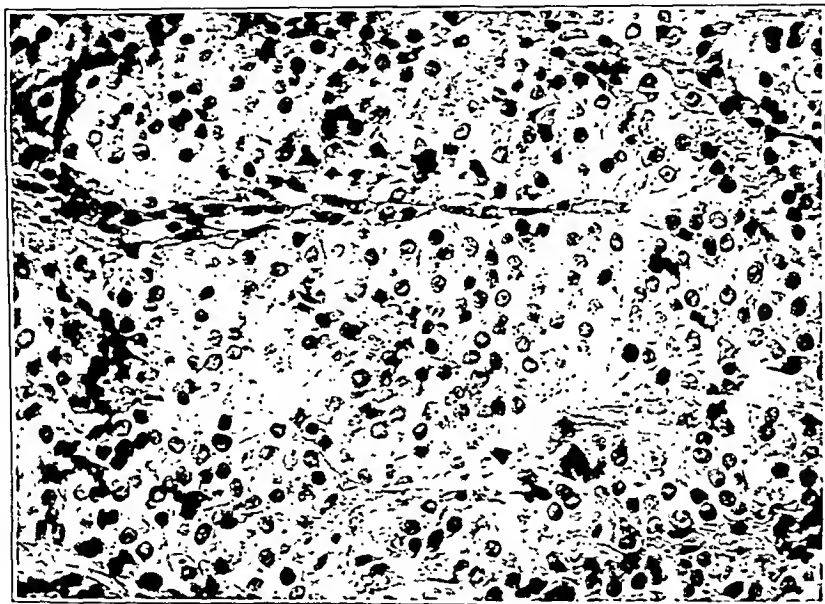


FIG. 232.—Case 3. Photomicrograph of chromophobe adenoma. (Hæmatoxylin and eosin stain.) ( $\times 300$ .)

**OPERATION.**—Dec. 26. Dr. Cushing exposed the bulging sellar floor by the usual transphenoidal route. The tumour had already burst its dural capsule and, covered by mucous membrane alone, was protruding into the sphenoidal sinus. This portion of the growth, together with the remaining fragments of the sellar floor and some of the intrasellar growth, was removed.

*Post-operative Course.*—There was a rapid and uneventful recovery. Definite improvement of visual acuity and a slight expansion of the visual fields were evident on Jan. 13, 1924, when he was discharged. One X-ray treatment was given fourteen days after the operation, and radiation at intervals of three weeks was advised. He reported on July 22, 1924, that he was feeling well and vision was slowly improving.

**PATHOLOGY.**—Sections show the tumour (*Fig. 232*) to be composed of elongated epithelial cells, with small oval nuclei. The cells are divided roughly into thick cell columns by small capillaries and occasional larger thin-walled blood sinuses. The connective tissue is practically confined to the walls of the blood-vessels. The cytoplasm of the cells contains no granules except mitochondria.

**DIAGNOSIS.**—Chromophobe adenoma.

The outstanding features of this very typical example of a pituitary tumour in a young adult are: (1) The neighbourhood symptoms of the growth, and particularly the loss of vision, which led to his admission to a surgical ward; (2) The constitutional signs of adiposity, hypotrichosis, somnolence, lowered basal metabolism, and so on. As anticipated, a chromophobe adenoma was disclosed.

The following case portrays the disease as it is frequently encountered in the middle-aged and elderly, who show what in this Clinic has long been designated as the wrinkled (and often lean) type of pituitary insufficiency.

*Case 4.*—*Surg. No. 19512.* Chromophobe adenoma of middle age with hypopituitarism. Operation. Improvement in vision.

ADMISSION.—Aug. 23, 1923. Mr. J. A. K., age 52, married, a bookkeeper of English birth, referred by Dr. D. H. Bell, of Tacoma, Washington, complaining of failing vision.

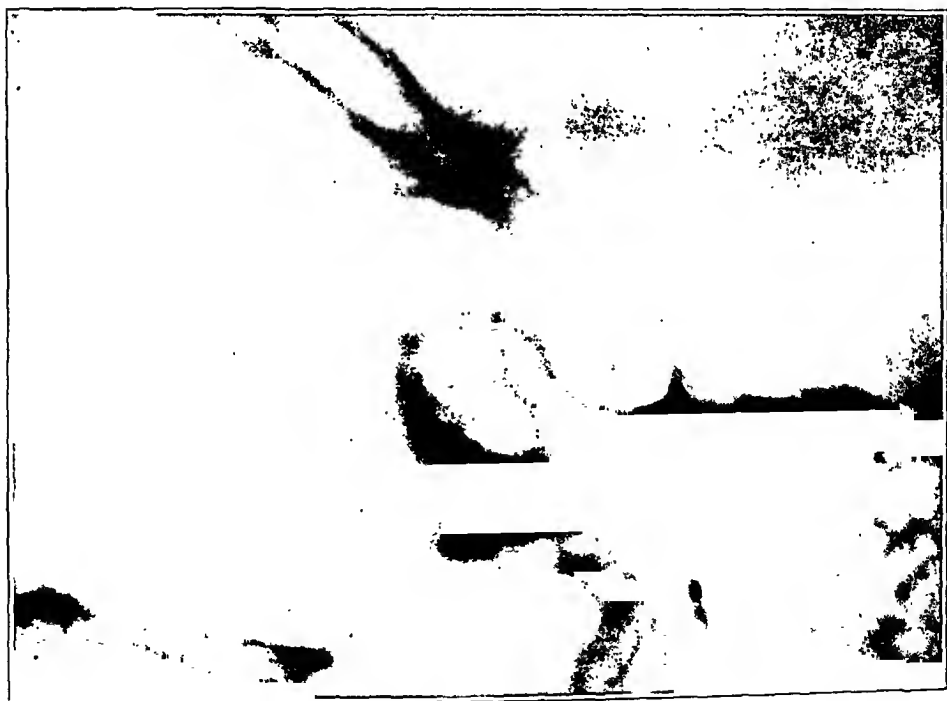


FIG. 233.—*Case 4.* To show the enlarged sella. ( $\times 1$ .)

CLINICAL HISTORY.—After a normal childhood he showed during youth a disinclination for rough outdoor sports. He has never had to shave more than two or three times in a month. He married; two children were born to him; the elder, a boy, was killed in the war; the younger, age 17, is a healthy girl. His wife is living and well. Two years ago he began to lose initiative and ambition in his work, and has become progressively more listless and apathetic. During this time he lost his *potentia sexualis*. Eighteen months ago his eyesight began to fail. His lateral fields of vision were subjectively the first to be lost. His left eye has been blind for the past two months, and vision for reading has been lost on the right. He



has had no headache, nor has he experienced unusual thirst or excessive frequency of urination. His weight has not altered appreciably during the past thirty years.

**EXAMINATION.**—The following positive findings were revealed: *Local.*—An advanced bilateral optic atrophy; V.O.S. light perception only; V.O.D. 5/200. Though he gives a clear history of bitemporal hemianopsia, the vision of the left eye is at present too poor to estimate its field; a right temporal hemianopsia is present. X rays show the sella to be distended, its floor depressed into the sphenoidal sinus, and its posterior clinoid processes absorbed (*Fig. 233*).

*General Features.*—He is a man of rather spare build; height 168 cm.; weight 64.4 kilo. Complexion is sallow; his skin is thin, soft in texture, very dry, and covered by fine wrinkles (*Fig. 234*), giving the impression of being withered and atrophic. On the scalp the hair is sparse, fine, and dry; it is very scanty on the face and pubes and in the axillæ; it is almost absent from the trunk and extremities.



FIG. 234.—Case 4. Photographs of patient with wrinkled type of skin.

The genitalia are small but normally developed. The basal metabolic rate is  $-4$  per cent. His fluid intake and output were normal under observation in the hospital.

**OPERATION.**—Aug. 30. Dr. Cushing operated, employing the customary transphenoidal procedure. The thin bony floor of the sella was removed and the dural capsule of the tumour was incised. Soft, adenomatous tissue under tension extruded itself through the incision. A portion of the growth was removed.

*Post-operative Course.*—Recovery was rapid and satisfactory, though with less prompt improvement in vision than usual. He was given a single X-ray treatment, and before his discharge on Sept. 19 some vision had returned to the left eye, to the extent of 5/100, while the acuity of the right had risen to 5/70. The perimeter showed at this time a complete bitemporal hemianopsia. In December he reported himself to be in good condition, and stated that a further improvement in vision had occurred, so that he was able to read the secondary headlines of the papers. He held this gain for nearly a year, when, according to reports, his vision again failed.

**PATHOLOGY.**—The tissue shows (*Fig. 235*) the tumour to be composed of elongated epithelial cells, divided roughly into thick cell columns by thin-walled blood sinuses. Connective tissue is scanty and confined to the walls of the blood-vessels. The cells have oval small nuclei and fairly heavy cytoplasm in which mitochondria are present but no other granules.

**DIAGNOSIS.**—Chromophobe adenoma.

The constitutional expression of the disease presented in this man differs from the usual manifestations chiefly in respect of the atrophic changes in the skin and hair, giving the patient a rather shrivelled look. This appearance, less characteristic of the hypopituitary syndrome than is marked adiposity,

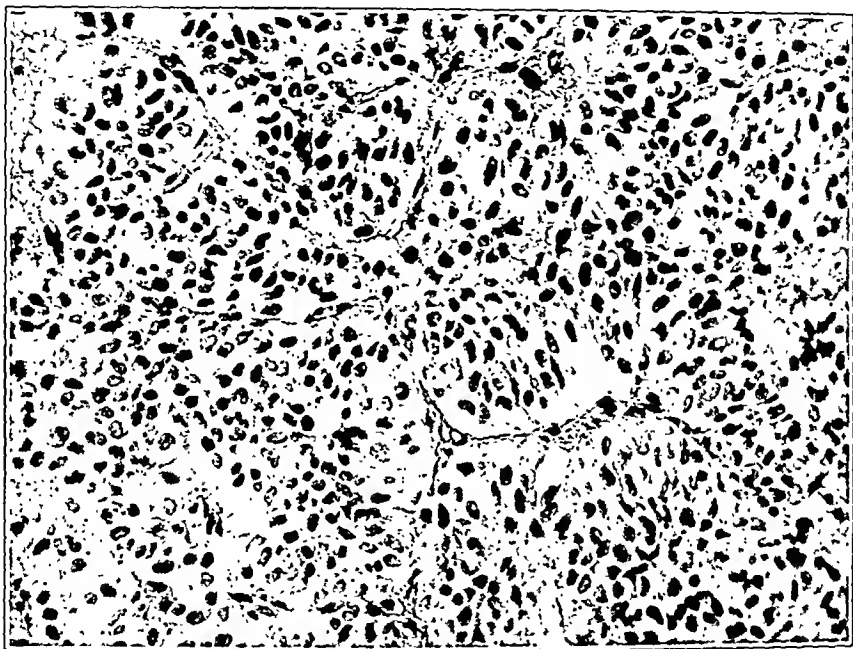


FIG. 235.—Case 4. Photomicrograph of chromophobe adenoma. (Hæmatoxylin and eosin stain.) ( $\times 300$ .)

is nevertheless not uncommon in patients past the prime of life. It may even be seen in young adults, to whom it gives a distinctly under-nourished if not emaciated appearance.

#### B. CHROMOPHIL ADENOMATA.

**Eosinophilic Adenoma with 'Hyperpituitary' Syndrome.**—Of the 162 cases, 39 were clinically acromegalic,<sup>19</sup> and in all but 4 an eosinophilic adenoma was identified. The tissues from the remaining four had been improperly fixed, but were in all likelihood of similar structure. The disease is essentially one of adult life. No case below the age of 20 entered the hospital. The numbers are evenly distributed in the third, fourth, and fifth decades of life. Only three cases were beyond the age of 50 at the time of admission. The disease appears to take a more chronic course than that

associated with a chromophobe adenoma. It is well known that the adenoma in many instances of acromegaly presents no surgical problem. The tumour may remain small and the patient may show no neighbourhood symptoms whatsoever. Indeed, widely ballooned sellae are less commonly seen in acromegaly than in hypopituitarism.

The average duration of recognizable acromegalic symptoms prior to admission has been six or seven years. The age incidence of onset is therefore considerably lower than the above figures indicate—a large proportion of cases beginning in the third decade of life, and a considerable number in the second decade. Gigantism, indicating that the disease had begun while growth was still in progress, was present in only four cases. The sexes are affected in approximately equal proportions, the males numbering 18 and the females 21. Below we present two examples of the well-known syndrome: (1) As it affects the adolescent; and (2) As it occurs in the young adult.

*Case 5.—Surg. No. 19847. An eosinophilic adenoma with gigantism. Radiotherapy, with untoward symptoms. Transphenoidal operation. Marked improvement in vision and general health.*

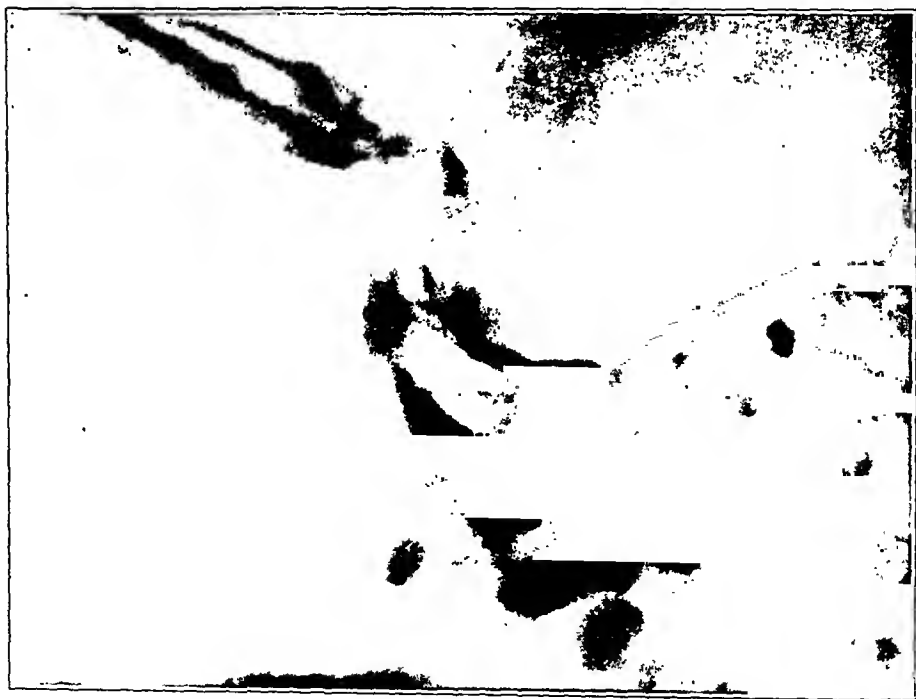


FIG. 236.—*Case 5. Showing enlarged sella in a case of gigantism. ( $\times 1$ .)*

**ADMISSION.**—Oct. 8, 1923. Mr. C. V. C., age 21, unmarried, referred by Dr. G. W. Crile, of Cleveland, Ohio, complaining of failure of vision, headache, and weakness.

**CLINICAL HISTORY.**—Early childhood was uneventful, except for the fact that at the age of 11 years he was struck on the right eye by a tennis ball—an event which

has left some permanent impairment of vision. About the age of 13 years he began to grow rapidly, and soon he towered above his schoolmates. He was not good at games, and, despite his unusual size, when compared to his fellows he was feeble and weak. It soon became apparent that his hands and feet were enlarging to a size out of proportion even to his great stature. He required shoes especially made to fit him.

Three years ago (in his eighteenth year) he had a temporary loss of vision in the temporal field of the left eye. This lasted about one month and then passed off spontaneously. For the past year he has suffered occasionally from mild frontal headaches. He has felt 'nervous' and easily tired. He noted that his hands and feet perspired excessively, and that his limbs were apt to become numb and to tingle if left for long in one position. Lack of ambition, feebleness, and lethargy have been progressive. He has attained the height of 6 ft. 8 in., but thinks he has ceased to grow within the past year. He has noted no unusual thirst nor undue frequency of urination.



FIG. 237.—Case 5. Photographs of patient.

**EXAMINATION.**—The following positive findings were revealed: *Local.*—There are some patches of old retinitis in the right fundus evidently due to his old injury otherwise the discs appear normal; V.O.S. 20/15; V.O.D. 20/30; visual fields normal, with the exception of two small paracentral scotomata in right field. The X-ray shows a widely distended sella (Fig. 236), its floor depressed into the sphenoidal sinus, and both anterior and posterior clinoid processes eroded.

*General.*—He is a veritable giant (Fig. 237), 201 cm. in height and weighing 108.7 kilo. He is heavily built with broad shoulders and expansive chest. Examining the skeletal architecture one notes the prominence of the supra-orbital ridges and the disproportionate enlargement of the facial bones compared with those of the cranium. X rays show a thickened cranial vault and very large maxillary, frontal, and mastoid air sinuses. The jaw is massive, but there is no mal-occlusion of the teeth. There is a moderate degree of pathological kyphosis of the upper dorsal spine. The bones of the limbs are enormous, heavy in build, and their prominences are exaggerated—features which are especially marked in the distal limb

segments. There is moderate hyperplasia of the subcutaneous connective tissues of the hands and feet, giving the fingers a rounded appearance, with well-marked pads on the palmar surfaces. The thickening in the fingers and dorsum of the hand is curiously soft and inelastic. The feet show similar changes. The cutaneous surface itself is not remarkable. The hair is not unusual in quality, quantity, or distribution, with the exception that the beard is scanty for his age. There is profuse sweating of the palms of the hands and soles of the feet. The basal metabolic rate is  $\div 5$  per cent. He is asthenic, but not drowsy. Libido sexualis has never been very strongly experienced. The genitalia appear normal. The fluid intake and output were normal during observation in the hospital.

In the absence of any definite impairment of vision from pressure involvement of the chiasm, radiation was advised. One X-ray treatment was given in hospital, and arrangements were made for them to be continued at intervals of three weeks at his home. He was discharged on Oct. 17.

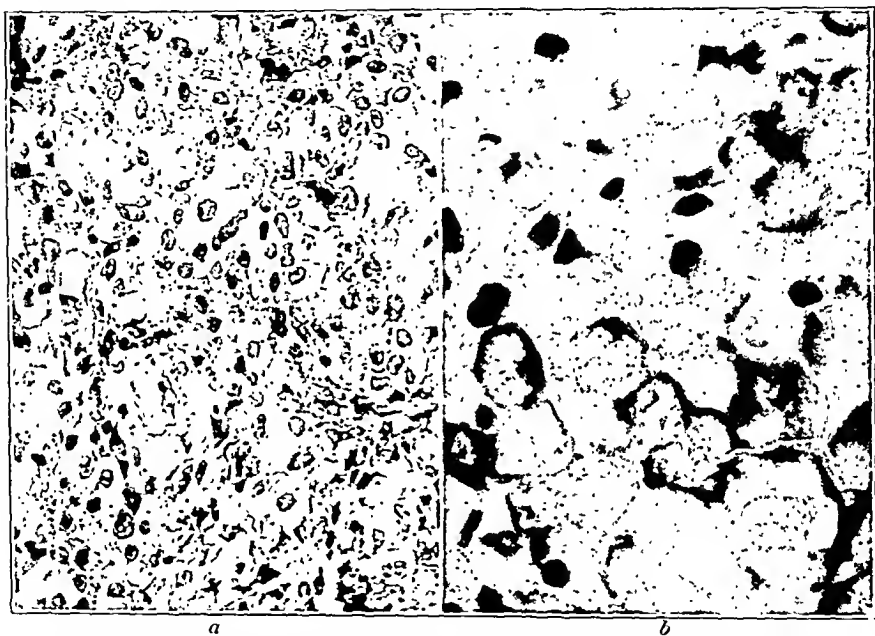


FIG. 238.—Case 5. Photomicrographs of chromophile adenoma. *a*. Hæmatoxylin and eosin stain. ( $\times 300$ .) After X-ray treatment. Note absence of connective-tissue septa. *b*. Neutral ethyl-violet-orange G stain. ( $\times 350$ .) Only eosinophilic granules and red blood-corpuscles appear black in photograph.

His general condition improved considerably during the next two months; his headaches were relieved and he felt stronger. However, on Dec. 18, a few hours after his fourth radiation (a prolonged treatment), he had a severe generalized headache, and felt sick and nauseated. He soon noted that his sight was failing and that the lateral field of vision of his left (the good) eye was gone, so that he could see only straight in front of him. He became seriously ill with prostrating headaches and an extraordinary degree of muscular enfeeblement, and on Jan. 11, 1924, was brought back to the hospital.

EXAMINATION.—At this time examination showed a distinct pallor of the left disc, with temporal hemianopsia; V.O.S.  $3/200$ ; V.O.D.  $20/30$ . The basal metabolic rate proved to be unaltered, remaining at  $\div 5$  per cent. The X rays showed no further change in the sellar outlines.

He was kept under observation in the belief that his exacerbation of symptoms was due to some acute degenerative process caused by the radiation and in the hope that his symptoms would spontaneously subside. However, the left eye gradually became almost blind and the acuity of the right fell to 20/50. His headaches meanwhile remained most severe. Surgical intervention was considered imperative to save vision.

**OPERATION.**—Feb. 2. Dr. Cushing by the usual transphenoidal approach exposed the 'paper-thin' sellar floor, and a generous decompression was effected, exposing the tense dura over the tumour. On incising the membrane a soft and almost liquid tissue began to exude. The adenoma was largely necrotic in its central part and much of it was scooped away.

**Post-operative Course.**—In spite of the fact that there was prompt relief from headaches and that within seven days his vision had greatly cleared and sight was returning in the left temporal field, it was long before his general condition of enfeeblement began to improve. Not until three weeks had passed did he regain sufficient vigour to be up and about. During his convalescence he had a remarkable period of polyphagia, being hardly satisfied with five meals a day. By the time of his discharge, Feb. 27, the vision had improved to V.O.S. 20/30, V.O.D. 20/30, and the left temporal field had opened out almost to its normal peripheries.

**Subsequent Course.**—Later reports state that he made an excellent recovery with unimpaired vision, that he has been free from headaches, and shows much more vigour than for some years past. His last letter, Feb. 2, 1925, tells of his leading an active life on a ranch in the West.

**PATHOLOGY.**—Sections of the tissue removed at operation show a mass of rounded cells of various sizes without architectural arrangement. Occasionally a thick-walled blood-vessel is seen. There are many multinucleated cells. The cytoplasm of the cells contains numerous fine granules which show a tendency to congregate in the periphery of the cells (*Fig. 238*).

**DIAGNOSIS.**—Chromophile adenoma.

The essential features of this unusual case are: excessive and disproportionate growth of the skeleton and somatic parts, hyperostoses, slight hyperplasia of the connective tissues, asthenia, depression of sexual function, hyperhydrosis, and so on, in connection with a large eosinophilic adenoma which had given no serious local symptoms until after excessive radiation. The circumstance that the disease afflicted this patient in adolescence makes the clinical picture predominantly that of gigantism. Particular attention may be drawn to the sudden increase of local symptoms following radiotherapy and their relief by operation.

For comparison with the preceding case, the following example of acromegaly has been selected, as the two patients were nearly of the same age on admission. In the acromegalic, however, the disease presumably began after growth had ceased.

**Case 6.**—*Surg. No. 20999. Eosinophilic adenoma with early acromegaly. Transphenoidal operation. Radiotherapy. Recovery without improvement in vision, but apparent abeyance of the disease.*

**ADMISSION.**—March 24, 1924. Joseph S., age 20 years, unmarried, a machinist, referred by Dr. W. D. Ayer, of Syracuse, New York, with the complaint of failing vision.

**CLINICAL HISTORY.**—He had developed quite normally until the age of 19 years. At that time he was a well-built, athletic youth, and had done well in school and at his work as machinist. Seven months ago he noted that his shoes—size 8, which size he had worn for some years—had become too tight for him. He observed also that a finger ring he was accustomed to wear had become too small and he was obliged to have it enlarged. His hands became so thickened and increased in



FIG. 239.—*Case 6.* Showing enlarged sella of acromegaly. ( $\times 1$ .)



FIG. 240.—*Case 6.* Photograph of young man with early acromegaly.

breadth that he was unable to get on his customary gloves. His friends soon remarked upon a change in his features, especially on the enlargement and thickening of the nose and lips. At about this same time his weight, which had remained fairly constant for several years, suddenly began to increase, and he has since gained 18.1 kilo. Coincidentally, his sexual libido disappeared and nocturnal emissions ceased.

Five months ago he had slight epistaxis, which recurred several times a week for about a month. Not long after this his vision began to fail (bitemporally) and has progressively grown worse. At the same time he began to suffer from recurring frontal headaches. These, however, have never been severe.

Four months ago he began to have frequency of urination during the day. At this time his doctor detected sugar in his urine, made a diagnosis of diabetes, and instituted diabetic treatment, which was maintained for about six weeks. His

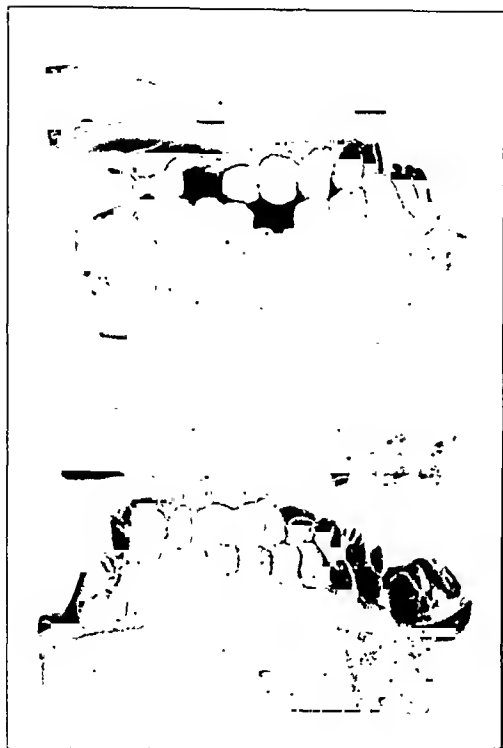
polyuria gradually subsided. Two months later, when all dietary precautions had been discontinued, no sugar could be detected in his urine. (As he had changed his doctor, exact information as to the course of his glycosuria is not obtainable.) He has noted occasional transient diplopia during the past two months. Within the past six weeks he has experienced on three or four occasions the sensation of a peculiar and disagreeable odour, which others could not detect.

**EXAMINATION.**—The following findings were revealed: *Local.*—Optic discs slightly hazy with veins moderately engorged; no definite pallor; V.O.S. light perception only; V.O.D. 20/200; a temporal hemianopsia is present on the right. X rays show the sella to be widely distended, though not markedly depressed into the sphenoidal cell (*Fig. 239*).

*General.*—A well-nourished young man (*Fig. 240*) who shows a marked tendency to somnolence, apathy, and disinterest. His height is 174.5 cm. His weight is 71.0 kilo. On examining the skeleton, one notes a marked prominence of the supra-orbital ridges, and the X rays show the frontal and maxillary sinuses to be unusually large; the lower jaw is much too wide for the upper, so that mal-occlusion of the molars is present (*Fig. 241*).

He is somewhat 'round-shouldered', though the curvature of the dorsal spine hardly amounts to a pathological kyphosis. The bones of the distal limb segments are disproportionately heavy and their prominences are accentuated. A skigram of the hand shows the bones of a heavy, squared type, with definite 'tufting' of the terminal phalanges.

There is a marked hyperplasia of the connective tissue throughout the body. The dermis and subcutaneous tissue are generally thickened and inelastic; this is especially apparent in the face. The nose and lips are thickened; the features are heavy and coarse; a deep, permanent furrow crosses the brow transversely; the facial tissues have lost their natural resiliency, and expression is therefore inadequate. The hands are similarly affected; the fingers thick and square-ended, with heavy pads of inelastic subcutaneous tissue; the whole hand is broadened and the skin



*FIG. 241.*—Case 6. Cast of teeth to show prognathism and mal-occlusion.



of the palm deeply furrowed. The subcutaneous connective tissue of the feet is likewise thickened; the toes are enlarged and clumsy and the feet broad; there are prominent post-calcanean pads. The tongue is enlarged; its motility is impaired by fibrous hyperplasia, so that the speech is thick and slurred. The hair is normal in quality, quantity, and distribution. There is profuse sweating of the palms and soles.

The blood-pressure is 118/80. The basal metabolic rate is +31 per cent. There is a definite history of glycosuria, but though he is at present on an unrestricted diet the glucose internal assimilation curve is normal. There is moderate enlargement of the heart; other viscera appear to be normal.

Two pre-operative X-ray treatments, directed to the hypophysis, were given without affecting the constricted visual field peripheries in any appreciable degree.

OPERATION.—April 5, 1924. Dr. Cushing exposed the bulging sellar floor by the customary transphenoidal route. After removing the flakes of thinned bone and incising the tense dural capsule, soft adenomatous tissue was disclosed, and as much of it as was deemed safe was scooped away.



FIG. 242.—Case 6. Showing change in configuration of hands (pre-operative left; post-operative right) after ten-months' interval.

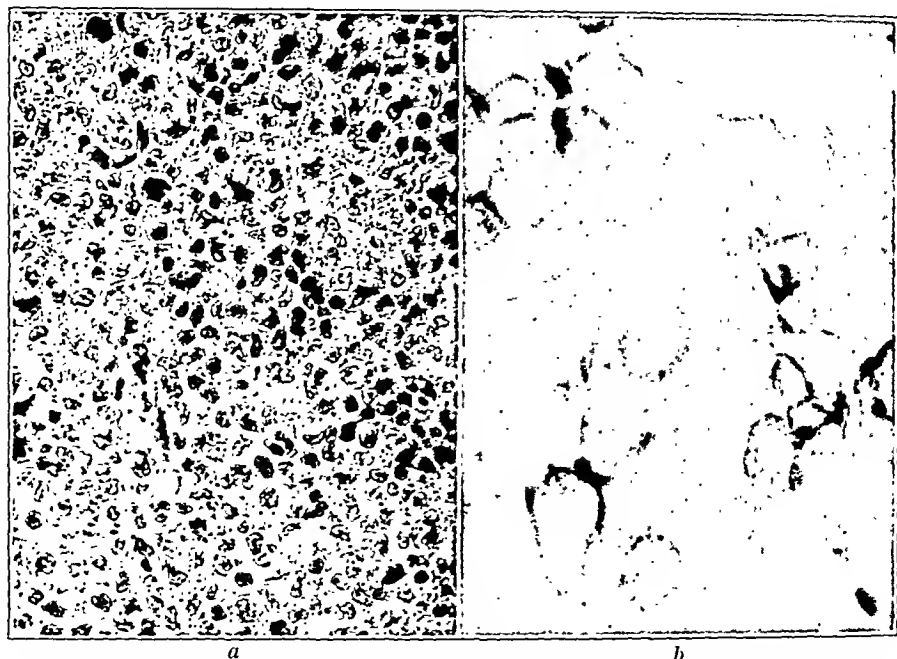
*Post-operative Course.*—A rapid and uneventful convalescence ensued. A third X-ray treatment was given before discharge. The headache, apathy, and somnolence were relieved. The visual acuity and fields showed no appreciable changes on his discharge on April 20; subsequent reports from his doctor recorded some improvement.

(The patient returned for examination on Feb. 25, 1925, ten months after his operation. He meanwhile had had thirteen X-ray treatments. There had been less improvement in vision than had been hoped for, and though the acuity had doubled on the right, the sharply cut hemianopsia persisted, and a restored patch of vision in the nasal field of the previously blind left eye did not help him greatly. But in other respects he had undergone an extraordinary change. This was chiefly noticeable in the subsidence of the pituitary 'myxoedema' or thickening of the soft parts. The change in his hands is shown by a comparison of the casts taken on the two admissions (Fig. 242); his shoes had become too large for him. There was, of course,

no change in the skeletal configuration, but he had lost about ten pounds in weight, his metabolism had gone down from  $+31$  to  $+13$  per cent, and his whole bearing was more alert and energetic.—P. B.)

**PATHOLOGY.**—The fragments of tissue are found to be composed of masses of polygonal cells (*Fig. 243*). They have no architectural arrangement. Blood-vessels are practically absent. The cells vary enormously in size, as do their nuclei. Almost a third of the cells have more than one nucleus, as many as six being present in some of them. The cytoplasm is packed with fine granules.

**DIAGNOSIS.**—Chromophile adenoma.



**FIG. 243.**—*Case 6.* Photomicrographs of tumour. *a.* Hematoxylin and eosin stain. ( $\times 300$ .) Note absence of connective tissue septa and presence of multinucleated cells. *b.* Neutral ethyl-violet-orange G stain. ( $\times 850$ .) Only eosinophilic granules appear black in photograph. Granules very fine, making a poor photomicrograph.

The leading characters of the disease presented by this patient, in addition to the definite signs of local pressure, are: progressive structural deformity of the skeleton; hyperostosis; hyperplasia of the connective tissue throughout the body; significant recent gain in weight; asthenia; depression of sexual function; raised basal metabolic rate; hyperhydrosis; temporary glycosuria. This is a typical example of the acromegalic syndrome in a young adult accompanying which an eosinophilic adenoma is found.

#### C. MIXED ADENOMATA.

**The Mixed Adenoma with 'Dyspituitary' Syndrome.**—It has long been known that symptoms of the acromegalic and hypopituitary syndromes may be variously combined in the same individual. The hypopituitary symptoms in these cases have been variously interpreted. Tamburini supposed that they were due to a toxic product elaborated by the hypophysial tumour.

Others have attributed them to the consequences of necrosis or hæmorrhage in the adenoma or to pressure of the tumour on the base of the brain. Cushing<sup>18</sup> has assumed that just as a long-standing case of hyperthyroidism may occasionally show traces of myxœdema, so in hyperpituitarism evidences of lowered function might be superimposed on the fixed characters of acromegaly. His address before the Harvey Society in 1910 ended with the following paragraph:—

It must be borne in mind, furthermore, that a hypophyseal struma (adenoma), which at one time has represented a condition of functional hyperplasia, may in the end actually block or destroy the secretory possibilities of the gland. As a matter of fact it may be expected: (1) That in all cases of original hyperpituitarism associated with tumour, the functional end-result will be hypopituitarism; and (2) That in many of the cases in which existing hypopituitarism is the striking feature traces at least of an early tendency to hyperpituitarism can be detected. These mixtures of the two syndromes of glandular over- and under-activity are most conveniently designated as dyspituitarism.

It should be pointed out, however, that, in many cases at least, the features of acromegaly and of the hypopituitary syndrome develop synchronously rather than in sequence. The distinct histology of the mixed adenoma, and the mixed nature of the associated glandular symptoms from an early stage of the disease, should be clearly recognized.

Some 13 of these mixed syndromes have occurred in the 162 cases that we have studied. The microscopic structure of the tumours found in these cases reflects the mixed character of the clinical syndrome. We will describe here one patient whom Dr. Cushing regarded as having a pure hypopituitary syndrome. Professor Biedl, who was visiting the clinic at the time, considered him to be acromegalic. The adenoma removed at operation in this case proved to be of mixed type.

*Case 7.—Surg., No. 19907. Adenoma with mixed syndrome. Transphenoidal operation. No improvement. Death six months later.*

**ADMISSION.**—Oct. 17, 1923. J. M., age 19, single, a Jewish factory worker, referred by Dr. H. H. Beers, of New York City, complaining of failure of vision and headache.

**CLINICAL HISTORY.**—His early development was uneventful. He is the smallest member of his family, of whom none are large. Puberty was delayed and did not occur until his eighteenth year—two years ago. At this time he had a few nocturnal emissions at intervals of two or three months. Sexual libido was never pronounced. Shortly after the onset of puberty, nocturnal emissions ceased and his sexual desires were entirely lost. He failed to acquire the secondary masculine distribution of hair.

One year ago he experienced tenderness to pressure on the right eye. Six months ago he began to suffer from recurrent, dull, aching pain behind the right eye, followed a month later by frontal headaches of moderate severity. Four months ago vision began to fail bitemporally, and it has rapidly deteriorated, so that now he can read only large headlines. He has experienced no unusual thirst or undue frequency of urination. There has been no notable alteration in weight during the past four years.

**EXAMINATION.**—The following objective findings were disclosed: *Local.*—A bilateral primary optic atrophy; V.O.S. 20/70; V.O.D. 20/200; bitemporal hemianopsia. The X rays show the sella turcica expanded, its floor depressed into the sphenoidal sinus, and its anterior and posterior clinoid processes eroded (*Fig. 244*).

*General.*—He is of average height, 171.3 cm., and weighs 64 kilo. His shoulders are narrow and his hips wide and rounded, resembling the feminine habitus (Fig. 243). The skin is very soft, fine, and smooth, like that of a child. The subcutaneous connective tissue of the lips, ears, and nose is thickened and inelastic, causing some immobility of these structures and giving to the features a certain coarseness. In the hands the subcutaneous connective tissue is thickened, forming well-marked pads on the palmar surface of the long fingers. The hair of the scalp is abundant, soft, and fine. He is quite beardless, and the axillary hair is absent. The pubic hair is scanty and is sharply limited above by a horizontal line and the genitalia are somewhat undersized. The basal metabolic rate is - 16 per cent. The fluid intake and output are normal during observation in the hospital. The urine is normal. A pre-operative X-ray treatment was given.



FIG. 244.—Case 7. Enlarged sella of mixed adenoma. ( $\times 1$ )

*OPERATION.*—Nov. 1. Dr. Cushing exposed the bulging and thinned sellar floor by the usual transphenoidal procedure. On incising the dura, soft, yellowish, oedematous tissue was exposed, and a considerable amount of it was spooned away.

*Post-operative Course.*—A rapid, uneventful recovery followed the operation. At date of discharge, Nov. 23, V.O.S. had improved to 20/30 while V.O.D. remained unaltered. A well-defined bitemporal hemianopsia remained. He still complained of mild, dull, aching pain behind the right eye. Two further X-ray treatments were given, and arrangements were made for them to be continued at intervals of about one month. Some weeks after his return home he had a series of convulsions in association with a local infection of the lip due to the retention of a piece of cotton-wool. He reported in person on March 21, 1924, his condition being practically unchanged. The results of the operation were therefore most disappointing, and



FIG. 243.—Case 7. Photograph of patient with 'mixed' syndrome.

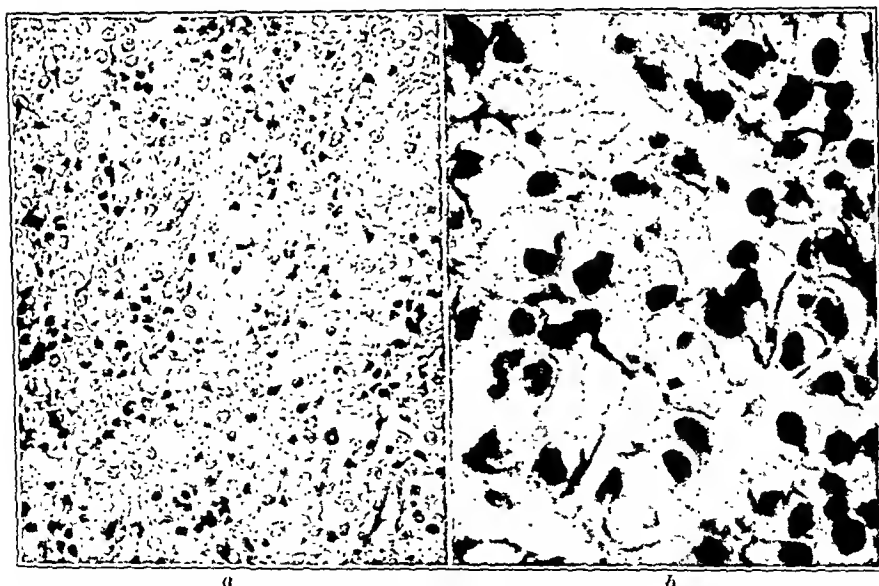


FIG. 246.—Case 7. Photomicrographs of tumour. *a*, Haematoxylin and eosin stain. ( $\times 300$ .) Note absence of connective-tissue septa. *b*, Neutral ethyl-violet-orange G stain. ( $\times 550$ .) Note ring of granules in periphery of cells.

its primary object in improving vision had failed. Six weeks later he suddenly lost consciousness, and had a severe hæmorrhage from mouth and nose, with fatality in a few hours. A post-mortem examination was not made.

**MICROSCOPIC EXAMINATION.**—On superficial examination the tissue removed at operation resembles a chromophobe adenoma. The cells are elongated, with small single nuclei, and connective-tissue septa carrying small blood-vessels are frequent. However, certain areas are without adenomatous arrangement, and a specific stain shows a ring of eosinophilic granules in the periphery of most of the cells (*see Fig. 223, D and Fig. 246*). It must, therefore, be classified as an adenoma of mixed type.

The outstanding characters in this case were : a mixed type hypophysial adenoma ; signs of local pressure ; depression of sexual function ; delayed puberty ; hypoplastic character of skin and hair ; slight skeletal build ; under-sized genitalia ; and a certain amount of connective-tissue hyperplasia giving suggestive evidences of overgrowth in lips, nose, and hands.

#### IV. THE SYMPTOMATOLOGY OF HYPOPHYSIAL ADENOMATA.

It is convenient to discuss the symptomatology of the adenomata under separate headings, for the reason that certain of the symptoms and signs may be associated with a pituitary tumour of any sort, while others fall into groups associated with specific types of adenoma. We shall discuss : (1) *The local pressure effects of the tumour* ; (2) *The signs of general intracranial tension to which it may give rise* ; (3) *The peculiar features of the hypopituitary syndrome* ; (4) *The constitutional disturbances of the acromegalic syndrome* ; (5) *The constitutional features of the mixed syndrome*.

**1. Local Pressure Signs.**—These are common to all pituitary adenomata which attain sufficient size to produce them.

**Headache.**—This, as Dr. Cushing has suggested, is in most cases due to stretching of the dural capsule. It is typically a dull, aching pain, or a sensation of pressure and discomfort, referred to one or both temples or behind the eyes. This discomfort is often an early symptom, and may have entirely subsided by the time the sella has become widely distended. When it persists, it is usually completely relieved by a sellar decompression.\* A subtemporal decompression, on the other hand, which usually relieves the headaches of other forms of intracranial tumour, has no effect on pituitary headaches.

**Roentgenological Signs pertaining to the Sella.**—For a satisfactory examination stereoscopic radiography should be employed. The sella which contains an adenoma is characteristically expanded by pressure from within—it is ‘ballooned’. Examining the sellar boundaries *seriatim* one notes : The anterior clinoid processes are characteristically thin and pointed, being scalloped out on their lower surfaces ; one process may be more affected than the other, thus giving an indication of the direction of extension of the tumour ; in long-standing cases the two processes may be separated by an

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\* This, perhaps, does not apply so certainly to the persistent headaches which sometimes accompany acromegaly and which must be due therefore to some cause other than capsular distention.

interval wider than is normal; they may be entirely obliterated by pressure atrophy. The floor of the sella is typically depressed into the sphenoidal cell; its outline is commonly clear, smooth, and thin; occasionally it may be rendered irregular by the presence of supporting septa in the sphenoidal cell; in long-standing cases it may be partially eroded by pressure atrophy. The dorsum sellæ and posterior clinoid processes are usually much thinned, but they maintain with the outline of the expanded sellar floor a continuous curve; in long-standing cases they may be entirely eroded by pressure atrophy or so nearly so that several exposures may be required before the thin remaining scale of bone is caught on edge by the rays and becomes visible on the film.

We wish to emphasize that the characteristic appearances are those of an even, more or less globular expansion of the sella. So typical of an hypophysial adenoma is this appearance that the great majority of them can be differentiated thereby from other lesions distorting the sella. Of these, the craniopharyngeal pouch cyst is perhaps the most common. As a rule these tumours are extra-sellar in origin, and though they distort the sellar outlines, the radiological picture is distinctive even in the absence of the customary shadows of calcareous deposits<sup>20</sup>. Moreover, these congenital cystic tumours usually give symptoms early in life, and only when one of them actually arises within the sella itself and remains free from calcareous depositions does the sellar deformation resemble that produced by an adenoma. Only two or three cases of this sort have occurred in Dr. Cushing's entire series. The sella may undergo pressure absorption from other conditions, from suprasellar meningiomata, for example, and more particularly as a late change when there is a marked and long-standing internal hydrocephalus; but experience should make it possible to distinguish the sellar changes produced by these lesions from those of the average adenoma unless the condition is so advanced as practically to obliterate all outlines of the fossa.

*Optic Changes.*—Distortion and compression of the chiasm and optic nerves, with a primary optic atrophy, is one of the early consequences of any hypophysial tumour. Later, if the neoplasm bursts through the capsule and reaches such proportions as to raise the intracranial tension by its actual bulk, which it rarely does, or by causing a secondary hydrocephalus—also a rare event—a definite 'choking' of the optic disc may be superimposed on the primary atrophy.

Projecting upwards, in the mid-line as a rule, the tumour implicates the chiasm in the first instance, and causes a bitemporal defect of the visual fields. The very typical manner in which a superior bitemporal notch first appears, and extends round the circle of each field, implicating in turn the upper temporal fields, the entire temporal fields, the lower nasal quadrants, and lastly the upper nasal quadrants, to total blindness, has been fully described by Cushing and Walker.<sup>21</sup> After the chiasm has been released from pressure, this process is exactly reversed (*Fig. 247*). The relationship of this sequence of events to the internal architecture of the optic nerves, chiasm, and tracts has also been fully discussed by these authors. While this is the usual type of visual disturbance, homonymous visual defects from lateral pressure on the chiasm, or impairment of one eye alone from implication

of one of the optic nerves, are not very rare; and innumerable combinations and varieties of these lesions may be encountered. The visual disturbances are the most frequent cause of the patient's seeking medical advice.

If the growth continues to enlarge it may reach enormous proportions, and in the course of the disease give rise to other local manifestations of pressure. Paresis or paralysis of the *oculomotor*, *abducent*, and *trochlear*

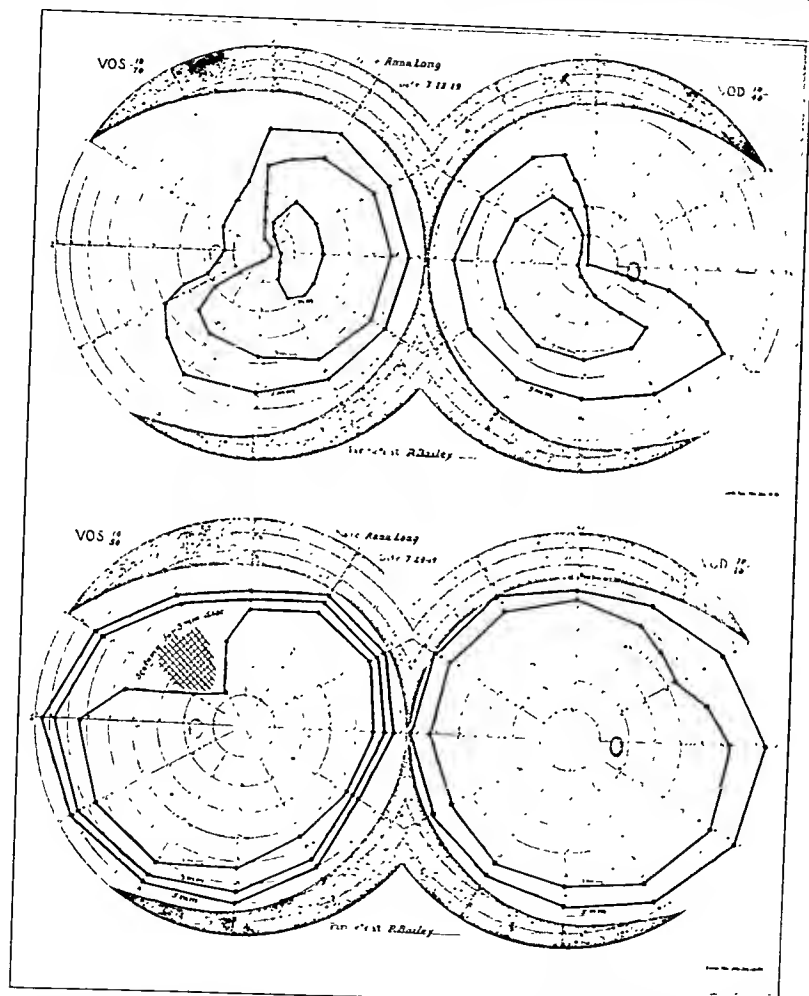


FIG. 247.—Visual fields to show improvement after transphenoidal operation. Pre-operative above; post-operative below. Operation performed July 19, 1919.

nerves may result therefrom. All combinations of paralytic strabismus, pupillary dilatation, and ptosis may be encountered in advanced cases. Occasionally the obstruction to the venous circulation from pressure on the cavernous sinuses causes œdema of the eyelids and obvious *congestion* of the venules of this region. A unilateral or bilateral *exophthalmos* may also result. These are late and inconstant evidences of a hypophysial tumour.



As the tumour distends the sellar floor and bulges into the sphenoidal sinus, congestion of the mucous membrane may cause *epistaxis*, but this is usually very small in quantity, merely staining the handkerchief from time to time. The deformity of the sphenoidal sinus may interfere with the proper evacuation of its secretions and a mild inflammatory condition of the mucous membrane may be induced, with retention and periodic evacuation of its contents, causing post-nasal dropping. There may be some consequent disturbance of the sense of smell, but *anosmia* from pressure implication of the olfactory nerve is comparatively rare. When present it is much less suggestive of an adenoma than of a meningioma, a form of tumour which has a predilection for the meninges of this region.

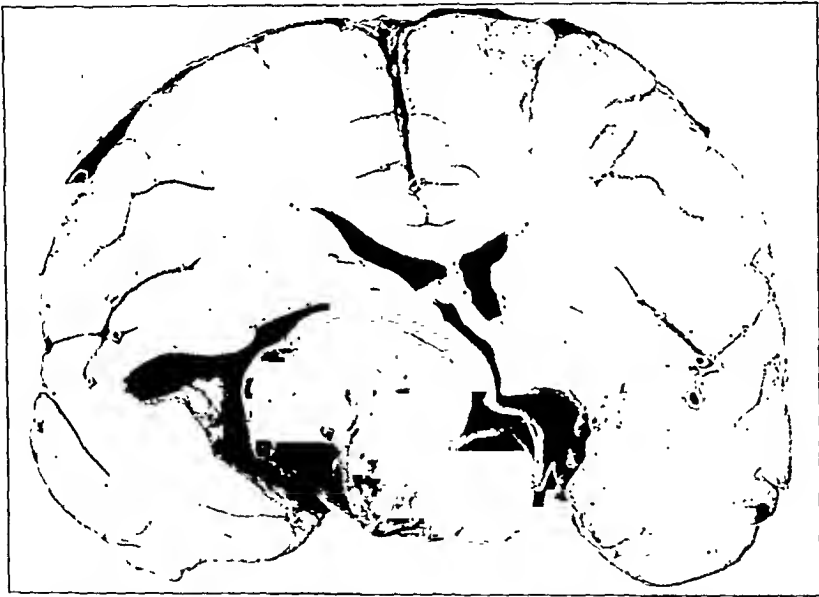


FIG. 24S.—Adenoma from a case of acromegaly. M. van W., *Surg.* No. 1784.  
Note the great distortion of the basal region of the brain.

Should the adenoma rupture its capsule and extend upwards into the cranial chamber in the median line, it may press upon and deform the hypothalamic region of the brain, and cause polyuria and polydipsia; but as a matter of fact these symptoms, which in exaggerated form constitute *diabetes insipidus*, are an infrequent consequence of pituitary adenoma. This disorder is to be sharply distinguished from the polyuria which occurs in association with the glycosuria of acromegaly, and which fluctuates with the sugar output just as in diabetes mellitus. As the uncinate region of the temporal lobe is subjected to pressure by lateral enlargement of the adenoma, typical *uncinate* attacks may occur—hallucinations of smell, usually of disagreeable odours; but this, too, is a rare symptom of hypophyseal adenomata.

In extreme cases (*see Fig. 24S*) the adenoma may protrude still further

into the cranial cavity, largely obliterating the cavity of the third ventricle and compressing the basal cerebral nuclei. *Drowsiness* and somnolence passing on to varying degrees of *coma* are common signs of such gross intracranial protrusions. The tumour may actually reach the level of the foramina of Munro and induce an acute obstructive hydrocephalus with general pressure symptoms.

**2. General Pressure Signs.**—Evidences of an increased intracranial tension are usually terminal features of hypophysial disease. They arise either on account of the actual bulk of the intracranial protrusion, or, as just mentioned, by virtue of a complicating obstructive hydrocephalus. Headache, vomiting, and choked disc superimposed on the already atrophic nerves are the leading signs, and their significance is peculiarly grave.

**3. The Constitutional Disturbances peculiar to the Hypopituitary Syndrome.**—

*Depression of Sexual Function.*—This is the first and practically constant symptom of the chromophobe adenoma, and is consistent with the fact that necropsies on these cases may reveal (apart from the hypophysial neoplasm) nothing of note except an atrophy of the gonads. Our most accurate index of its onset is amenorrhœa. Depression of libido sexualis, especially in the celibate male, may not be very apparent to the patient himself, though in a large proportion of our male cases loss of libido is one of the early symptoms to be recorded. In women, however, the cessation of the menses invariably attracts the patient's attention, and the constancy of amenorrhœa as the earliest symptom is remarkable.

Of all the female cases between the ages of 20 and 40 years in the series, there are but two exceptions to this rule. One of these was complicated by the presence of hyperthyroidism, and the basal metabolic rate was +22 per cent. Even in this woman, age 32 years, the menses had been irregular and scanty since their onset at the age of 16 years, and complete amenorrhœa developed shortly after the appearance of the local manifestations of hypophysial tumour. Whether the co-existence of hyperthyroidism had any influence in maintaining menstruation, we cannot tell. In the other exception to which reference has been made, a woman of 31 years, menstruation remained regular with but two lapses—once in 1916 and again in 1922, when a single menstrual period was three weeks late in its appearance, though local signs of hypophysial tumour had been present for many years. Following the transphenoidal operation for the relief of local pressure signs complete amenorrhœa developed.

It is characteristic of the amenorrhœa of the hypopituitary syndrome that it is of gradual onset. Complete amenorrhœa is usually preceded by a variable period of irregularity and paucity of the flow. Operative intervention, which usually consists in partial removal of the tumour combined with a sellar decompression, generally has no influence upon the existent depression of sexual function. In the series there is but one notable exception to this rule. A woman, age 27, with typical amenorrhœa and other hypophysial manifestations of a typical chromophobe adenoma, was operated upon by the usual transphenoidal procedure and was put on a course of glandular (anterior lobe) therapy. Five months later, regular menstruation returned.

Six months thereafter, she became pregnant, and in due course was delivered of a normal child.

*Atrophic Changes in the Skin and Hair.*—Atrophic changes in the skin and hair are, in our own experience, as constant signs of the chromophobe adenoma as is sexual depression. They are minor changes and occasion no inconvenience to the patient, and they must be carefully sought by the observer. For these reasons any attempt at a statistical review of them would be unreliable and fruitless, and no opinion can be formed of the time of onset of these changes relative to other symptoms. In our personal experience we have encountered but one case in which they were entirely absent (cf. *Case 1*). Their character varies somewhat with the age of the patient. In the adolescent the skin is like that of a little child—thin, delicate, smooth, and practically hairless. In the adult the skin is again thin, delicate, smooth, and usually unduly dry. The hair of the head is of normal quantity, but of a fine, soft, dry texture. The facial, axillary, and pubic hair is apt to be scanty, and the distribution of the latter is of the feminine type. Hair is much diminished or absent over the remainder of the trunk and over the limbs. From the age of about 35 onwards, the skin, which is still soft, thin, and delicate, becomes finely wrinkled. This fine wrinkling is a very characteristic appearance; it is well shown in *Fig. 234*. At all ages there is a very definite lack of the fibrous tissues of the cutis, secretion of sweat is diminished, and the hair is scanty and atrophic.

*Adiposity.*—This is a fairly common symptom in the hypopituitary syndrome. Among the younger patients it is definitely present in at least 80 per cent of the cases; but it may be entirely absent. Among the older patients it is less manifest, being present in about 50 per cent. Apart from actual pathological adiposity at the time of examination, previous unaccountable fluctuations in the patient's weight are of significance. In patients showing no particular obesity at the time of examination, it is quite common to obtain a history that within the past few years there had been a sudden gain of some 10 or 15 kilo., which was subsequently lost. Except for the unusual pallor of the skin there is nothing particularly characteristic about the adiposity of pituitary disease to distinguish it from adiposity due to other causes; but the history of sudden fluctuations in weight, whether maintained or not, is very typical. Unlike other victims of adiposity, these people are apt to suffer from the cold.

*The Basal Metabolic Rate.*—This is usually low in the hypopituitary syndrome. It is never above normal. It varies between about  $-5$  per cent and  $-35$  per cent from normal. We do not consider a reading which falls within 10 per cent of the normal standard to be of pathological significance, and a considerable number of cases come within this limit. The average, however, is about 20 per cent below normal. The low basal metabolic rate, no doubt, accounts for the lethargy and inactivity with slow pulse and slightly subnormal temperature which the majority of these patients exhibit.

*Infantilism.*—Infantile characters are not typical of hypophysial adenomata, for the reason that the tumour is very rare during the pre-adolescent period of life. In two cases (cf. *Case 2*) in which there was a suggestion of infantilism, it was of slight degree, because the patients were well advanced

toward adolescence when the disease affected them. We know of no recorded case of hypophysial adenoma in which infantilism was a striking feature.\* This is in marked contrast to the syndrome accompanying the craniopharyngeal pouch cyst of developmental origin, which usually manifests itself in childhood and is associated with a very striking degree of infantilism.

*Carbohydrate Tolerance.*—Since attention was first drawn to the subject by Goetsch, Cushing, and Jacobson,<sup>22</sup> much has been written on carbohydrate tolerance as affected by hypophysial disease, especially in those types with which we are now dealing. Before the days of clinical calorimetry, a study of sugar tolerance was about the nearest approach one had toward an estimate of the metabolism of these patients. Individuals were said to have 'a high or low carbohydrate tolerance', depending upon the amount of glucose they could ingest without manifesting glycosuria. It was assumed when tolerance was high, as is usually the case in hypopituitarism, that the patients possessed an abnormal facility in transporting, modifying, and storing glucose in their bodies. In order to avoid the uncertain factor of alimentary absorption in this test, we have carried out intravenous administration of glucose in a number of cases. This we term the internal glucose assimilation test.

Contrary to the former belief in a 'high glucose tolerance', we find that the blood-sugar curve is considerably longer in returning to its basal level in these individuals than in normal persons—i.e., their capacity for dealing with glucose in the blood-stream is subnormal. One can only conclude from this observation that the alimentary test, although useful as a clinical investigative procedure in hypopituitarism, has proved misleading from a physiological standpoint. It appears probable that absorption from the alimentary canal is so slow in them that even their retarded biochemical mechanism can keep the blood-sugar level below the 'kidney threshold' for glycosuria. It is quite probable that the retardation of both processes—alimentary absorption and internal distribution—merely reflects the lowered metabolic rate which we know these patients have. Naturally, in connection with the hypopituitary syndrome glycosuria never occurs—in contrast, as we shall see, to the findings in acromegaly.

#### 4. Constitutional Disturbances peculiar to the Acromegalic Syndrome.—

*Depression of Sexual Function.*—It will be noted that depression of sexual function is a symptom common both to the hypopituitary and acromegalic syndromes. On the average it is probably one of the earliest symptoms in both disorders, but the histories of the acromegalics record many exceptions, and in some cases menstruation and libido sexualis have persisted for many years in the presence of a well-marked acromegalic symptomatology.

*Connective-tissue Hyperplasia.*—Hyperplasia of the connective tissue of the body is a constant and essential element of the disease. It is especially evident in the subcutaneous tissues of the lips, nose, scalp, hands, and feet, which become markedly thickened and their consistence stiff and inelastic. The dermis itself is thickened, and gives to the skin a coarse appearance and texture which is associated with considerable oedema. The visceral enlargement that is present in advanced cases is due to hyperplasia of their

\* Cf. footnote, p. 330.

# Symptoms etc.

## Local pressure.

- 1 Headache 2. X-ray (a) "Ball sound" (b) thin & pointed ant. clin. proc.
- (c) floor depressed, smooth & thin (d) post wall & clin. proc. flattened.
- (e) erosion of optic. (a) Primary optic atrophy. (b) bilateral defects, chiasma. (c) homonymous defects, lateral pressure (d) one eye alone, optic nerve. (e) paresis or paralysis of 3, 4 or 6 nerves.
- (f) venous congestion (g) Exophthalmos (h) epistaxis. (i) polyuria
- (j) polydipsia (k) small disorders (l) drowsiness, coma and obstructive hydrocephalus.

## General pressure (terminal)

- 1 Headache (2) Vomiting (3) Shattered disc.

## Symptoms peculiar to syndrome

<u>Symptom.</u>	<u>Hypopituitary.</u>	<u>Hyperpituitary.</u>
Depression Sex. function.	Slow irregular onset of amenorrhoea. Not altered operation.	Earliest symptom but some exceptions.
Changes skin and hair.	Atrophic - thin, delicate, smooth dry skin. Face and body hair scanty and of feminine type. After 35 fine wrink. Res.	Hypertrophy connective tissue, coarse sweaty skin. Hypertrichosis. "Bull dog scalp". Tiger pads.
Obesity various changes.	Fairly common. Sudden changes.	—
B.M. R.	-5 to -35%	Outstanding, face, armpits, jaw. Generally raised.
Carb. tolerance.	? High.	Low with variable glycosuria
Infantilism	Rarely. More marked in congenital pouch cyst.	—

## Pathological Anatomy

of  
hypoph. adenomata.

- A. Chromophobe, glandular insufficiency.
- B. Chromophil, two types (a) Eosinophilic = hyperpituitary syndrome (b) Basophilic, no clinical type.
- C. Mixed D Malignant



connective-tissue elements. The neuritic pains from which these patients suffer are probably due to the fibrous hyperplasia which is present in the nerve sheaths as well as to the narrowing of the intervertebral foramina from hyperostosis. The large, stiff, clumsy tongue of acromegaly is another example of fibrosis.

*Changes in Skin and Hair.*—As just mentioned, the skin becomes thick and coarse from fibrous changes. In certain parts of the body, such as the palms of the hands and the scalp, it may be deeply corrugated, giving occasionally a very remarkable appearance upon the scalp, which Dr. Cushing speaks of as "the bull-dog scalp of acromegaly" (Fig. 249). There is apt to be profuse sweating, often accompanied by a disagreeable odour. Hypertrichosis occurs sometimes to a remarkable degree, though this is by no means constant. All this is in marked contrast to the delicate, dry skin which accompanies the hypopituitary syndrome.

*The Osseous Changes.*—These are the outstanding features of the disease. The skeletal deformities tend to increase as time goes on, and of course remain permanent, even should the period of hyperpituitarism lapse. Hence, so far as these manifestations of the disease are concerned, the expression "once an acromegalic, always an acromegalic" applies. We need but mention the well-known enlargement and deformities of the bones of the face, the huge air sinuses, the undershot jaw with its wide angle; the upper dorsal kyphosis; the enlarged clavicles and ribs; the hyperostosis of the terminal phalanges of the fingers, and of the limb bones and vertebral column.

*The Basal Metabolic Rate.*—This is much less constant in acromegaly than in the hypopituitary syndrome. As a rule it tends to be elevated in the active periods of the disease, as in the case recorded (Case 6), and though registrations of  $\div 30$  per cent are not infrequent, it never attains the high figures seen in extreme cases of hyperthyroidism. On the contrary, normal or subnormal registrations are not uncommon.

*Glycosuria and Carbohydrate Tolerance.*—About 20 per cent of the acromegalics in the series have shown spontaneous glycosuria in greater or lesser degree. The glycosuria is characterized by its extreme variability from time to time in the same individual. At one period—usually after the disease is well established—it may be a prominent symptom, accompanied



FIG. 249.—To show the furrowed scalp sometimes seen in acromegaly. A. C., Surg. No. 4215.

by polyphagia, polyuria, and loss of weight. A few weeks later both it and its associated symptoms may have entirely disappeared. It is a common experience, with cases of acromegaly which are under observation, to discover spontaneous glycosuria on one or two isolated occasions. The glycosuria of acromegaly is certainly of a diabetic nature—i.e., it is due to a failure to utilize glucose in the body—but whether it is actually pancreatic in origin is undetermined. In this clinic one case died in typical diabetic coma, and glycogen was identified in the liver post mortem, in approximately normal amount.

We have not had the opportunity personally to investigate the 'carbohydrate tolerance' of an acromegalic who had glycosuria, but it is presumably low. In several who had not glycosuria the 'internal glucose assimilation curve' was within normal limits.

**5. The Constitutional Features of the Mixed Syndrome.**—Though the patients of this category present no symptoms other than those mentioned above in the acromegalic and hypopituitary syndromes, the symptoms of the two disorders are variously combined. The fat, hypotrichotic individual who at the same time has enlarged accessory nasal sinuses, some exaggeration of the supra-orbital ridges and of the various prominences of the limb bones, and is perhaps overgrown, is typical of the mixed class. It should be clearly understood that many cases which, on account of a marked preponderance of one or the other syndrome, are commonly relegated to that particular class, may yet be tainted by traces of the other. For example, the patient portrayed in *Case 5* whom we selected to typify acromegalic gigantism was almost beardless at the age of 21.

## V. PHYSIOLOGICAL CONSIDERATIONS.

To construct physiological theories from pathological findings is always hazardous, and it is especially so in regard to pituitary disorders, but so much stress has been laid in the preceding sections on the distinction between eosinophilic and chromophobe adenomata, and the correlation which exists between them and the clinical picture, that we are obliged to discuss the implications of this distinction, especially since we are dealing with a tumour of a glandular organ.

Contrary to the opinion of Sjövall<sup>23</sup> and others, the symptoms of pituitary disease have been, and must continue to be, interpreted in the light of animal experimentation rather than by deduction from the pathological findings in clinical cases, and especially is this true in cases of tumour and hydrocephalus, as one of us has already pointed out,<sup>24</sup> where the normal anatomical relations are enormously distorted. However, certain definite suggestions emerge from the clinical pathology. We shall consider the problem illuminated from this source alone in the first place. We shall then consider it in the light of both clinical pathology and experimental evidence.

From the facts furnished by pathological states, we should be led to consider that the normal hypophysis may be concerned with growth, with sexual function, with the condition of the skin and hair, and with the basal metabolic rate, since changes in these almost invariably follow upon destructive compression of the hypophysis by an adenoma (or other neighbouring



tumour). Nevertheless, we are faced with certain exceptions which require explanation; and we can obtain little evidence as to which part of the hypophysis is concerned. We should also surmise that adiposity, hyperplastic changes in the bones and connective tissues, polyuria, and glycosuria were due to some cause other than compression or destruction of the hypophysis, as they are present in only a variable proportion of cases.

Such conclusions, however, require careful investigation. We realize at once that, in addition to destruction of the pituitary gland, a complicating factor exists in that an adenomatous neoplasm is present which may or may not furnish a normal or a perverted hypophyseal secretion.

Let us first eliminate from the category of possible manifestations of hypophyseal destruction the symptoms which there is reason to believe are due to another cause. The hyperostoses and connective-tissue changes of acromegaly are constantly associated with an eosinophilic adenoma; they never accompany an anatomically similar tumour which is not eosinophilic; they are associated with no other constant pathological lesions. Acromegalic gigantism is dependent on the eosinophilic adenoma for the same reasons, in those cases in which the development of the eosinophilic adenoma has preceded the cessation of growth of the individual. Other factors, such as the site, size, and local effects of an adenoma being equal, the 'eosinophilia' of the tumour remains alone to account for these symptoms of acromegaly and of gigantism.

Let us now consider if any symptoms can be etiologically attached to a destructive lesion of the hypophysis from the evidence of clinical pathology. Depression of sexual function is a feature almost constant for all types of pituitary adenoma, and as it occurs so early as to preclude the factor of hypothalamic injury as a cause, we are left with two possibilities: (1) That all forms of adenoma produce some 'toxin' capable of depressing sexual function; (2) That destruction of the hypophysis or diminution of its secretion is responsible. Exceptions to the rule that depression of sexual function is the first symptom have occurred in but two cases of chromophobe adenomata. On the other hand, with eosinophilic adenomata early amenorrhœa is less constant. The only symptoms, however, which may precede amenorrhœa are the acromegalic changes in the bones and connective tissues, which, as we have seen, are probably due to the active secretions of the tumour, and are certainly not the consequences of local pressure of the tumour upon the hypophysis. In these cases amenorrhœa constantly precedes signs of local pressure, such as optic atrophy, just as it does in the cases of chromophobe adenomata. We must therefore conclude that an eosinophilic adenoma may induce the constitutional changes of acromegaly, by its secretory activity, before it has attained a size sufficient to cause depression of the function of the hypophysis by pressure. This further indicates that amenorrhœa is probably an early consequence of tumour pressure—that it is due to depression of the function of the pituitary gland, and is not a result of secretory activity nor of toxin production by the neoplasm.

We are unable to apply to the cutaneous changes of the hypopituitary syndrome the same line of argument as in the case of amenorrhœa, because it is impossible to determine accurately their time of onset. They are a

practically constant accompaniment of the chromophobe adenoma, and rarely occur with the eosinophilic tumour, which, on the contrary, causes excessive growth of hair and thickening of the skin. This suggests that the cutaneous changes of the hypopituitary syndrome are due to a loss of function, while the eosinophilic tumour prevents these effects of pituitary lesion by producing acromegalic changes by means of its own secretory activity.

The basal metabolic rate is constantly lowered with chromophobe adenomata, while it is often raised with eosinophilic adenomata. It is possible that the same line of reasoning applies as in the case of cutaneous changes.

Concerning the remainder of the clinical symptoms we can say little from the evidence derived from clinical pathology. It should also be noted that little concerning the functions of the individual parts of the hypophysis can be learned from this source.

So far we have restricted ourselves to the clinical pathology of hypophysial adenomata as an approach to the elusive physiology of the pituitary gland. A more fruitful and a surer way exists in the application of the evidence furnished by experimental research to a critical interpretation of the clinical data. It would be out of place to enter here into a full discussion of the experimental work bearing on pituitary physiology, but we may recapitulate our present views. Some time ago one of us, in approaching the problem from the experimental standpoint, recorded the following conclusions:<sup>24</sup> "(1) The pars anterior (pars distalis, vorderlappen) of the hypophysis is in its histological structure glandular. Its secretory product has not been isolated, and the effects which it exerts on the organism are not conclusively demonstrated; (2) The pars posterior (pars intermedia and pars nervosa) is structurally not glandular (in dog and man). Its removal from the organism (dog) gives rise to no symptoms". In the light of experimental evidence obtained since these conclusions were recorded the first requires amendment. It had already been shown by Fichera, Cushing, Aschner, and others that removal of a large part of the gland, including the anterior lobe in a growing animal, resulted in dwarfism. Smith and Graesser<sup>25</sup> have shown also that in the rat the pars distalis exerts a definite influence upon growth, and that extensive injury of the pars anterior results in dwarfism and infantilism, which can be overcome by the intraperitoneal administration of extracts of the pars distalis of the beef hypophysis. It is highly probable, therefore, that the rare infantilism and dwarfism occurring with chromophobe adenomata (and much more often with the suprasellar tumours) is due to depression of function of the pars distalis of the hypophysis.

This brings us to an interesting point which may elucidate some of the more obscure symptomatology. The eosinophilic adenoma, in spite of the fact that it depresses or eliminates the function of the normal hypophysis, yet induces gigantism when acting within the growth period. Here we have an example of a tumour interfering with an important physiological function, and in all probability substituting for that function the effects of its own secretory activity, which must be very similar to those of the normal gland. In other words, dwarfism should result from hypophysial destruction; but instead, the activity of the eosinophilic tumour prevents this effect and causes gigantism. Evans and Long<sup>17</sup> have produced gigantism in rats by suitable

administration of pars distalis preparations, and in this respect the effects of the eosinophilic adenoma are at least akin to the experimental hyperpituitarism thus produced. It is consistent to link with this similarity in biological activity the close histological resemblance of the eosinophilic tumour to the normal gland. We would further deduce from these premises that the eosinophilic granules of the normal gland are probably concerned with the growth of the organism.

When, however, we attempt to correlate acromegalic changes (as contrasted with simple gigantism) with experimental physiological findings we meet with difficulties, for acromegaly has as yet no experimental counterpart. In the present state of our knowledge, it is profitless to speculate upon the possibility as to whether the eosinophilic adenoma produces an excessive or a perverted secretion to account for the changes of acromegaly.

A word may be added concerning the pars posterior of the hypophysis. No physiological activity has been demonstrated from it in mammals, either experimentally or clinically, though numerous pharmacological activities have been obtained from its extracts. Its total ablation in the dog causes no symptoms, as many investigators have shown, and we suspect that its compression by a tumour in man causes no symptoms. At any rate, it probably plays no part in the symptomatology of the hypophyseal tumours which we are considering.

Polyuria and polydipsia are so rarely seen with hypophyseal adenomata, except for the polyuria accompanying the diabetes mellitus occasionally occurring in acromegaly, that they need not be discussed, except to note that if the essential lesion of diabetes insipidus be considered to lie in the pars posterior of the hypophysis, it is strange that it occurs so rarely with an intrasellar tumour such as the hypophyseal adenoma.

## VI. DIFFERENTIAL DIAGNOSIS.

The recognition of a tumour of the hypophyseal region presents little difficulty as a rule. The primary optic atrophy, characteristic visual field defects, the ocular palsies, the X-ray picture of sellar deformity, leave little room for doubt. It is essential, however, to carry the diagnosis a stage further, and to be able to form an opinion as to whether the tumour is or is not an adenoma, since from a surgical standpoint the correct differentiation of the adenomata from the various parahypophyseal lesions is of the first importance.

The eosinophilic adenoma is not likely to be confused with any other lesion, as the signs of acromegaly or gigantism with which it is associated are almost unmistakable. The chromophobe tumour, on the other hand, may be easily confused with a number of parahypophyseal lesions, since simple compression of the pituitary gland and neighbouring nerves are common to all of them. The more important of them are the suprasellar cysts originating from Rathke's pouch, suprasellar endotheliomata, teratomata, cholesteatomata, gliomata of the optic chiasm, and even hydrocephalus. Each of these conditions may be associated with the hypopituitary syndrome, probably by virtue of the common factor of compression of the pituitary gland. They

From an experience of 53 cases at this Clinic, in which between four and eight 'series' of treatments were given, it appears certain that the growth of the tumour may usually be retarded, and an actual reduction in its size sometimes effected, by radiotherapy. It should be mentioned, however, that the treatment is not entirely free from risk (cf. *Case 5*). In two of the 53 cases temporary aggravation of their symptoms was observed. Sudden and severe headache followed the treatments, they suffered from increased difficulty of vision, and from general lassitude and prostration. One of them was subsequently completely relieved by a sellar decompression operation. In the other the symptoms subsided spontaneously forty-eight hours after treatment, apparently consequent on a rupture of the tumour into the sphenoidal sinus. These temporary aggravations of the symptoms are presumably due to acute degenerative processes, accompanied by swelling of the tumour tissue. It may be said, however, that no histological change in the adenoma has been demonstrable in cases in which tissues have been secured by operation subsequent to periods of radiotherapy.

The experience in the Clinic with radium, so highly recommended by Hirsch, has been far less encouraging. In a number of cases, seeds of radium emanations have been implanted directly in the tumour after its exposure by operation. In other cases radium has been used by intranasal applications without operation. No beneficial results have been seen, and the occasional necrosis of bone resulting from these applications has been a distressing complication. We have had no experience with the extracranial heavily-filtered radium-pack method as advocated by Pancoast.<sup>23</sup>

**Medicinal Treatment.**—As regards endocrine substitution therapy, we are well-nigh powerless at present. The oral administration of 'whole gland' or of separate pituitary lobe preparations has proved quite valueless in the past. So convincingly true has this been that such treatment of hypophyseal disease was abandoned many years ago at this Clinic. However, the recent work of Evans holds out fresh hope in this direction. By intraperitoneal administration of anterior lobe preparations to rats whose hypophysis had been injured, his co-workers were able to overcome the retardation of growth from which the animals suffered. It is possible that some day we may be in possession of endocrine substitution therapy for hypophyseal disease quite comparable with that which is available for hypothyroidism, but it appears that it will have to be given in some other way than by mouth.\*

**Surgical Treatment.**—What can we hope to achieve by mechanical measures? In the first place it must be recognized that from the position of the tumour, its intimate relationships to important structures, and its mode of extension around these structures, its complete removal is surgically impossible, even if the attempt were advisable, in the great majority of advanced cases. Moreover, we believe that any operation which aims at

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\* There is at present no justification for calling the administration of pituitrin for diabetes insipidus an endocrine substitution therapy. In fact, the experimental and clinical evidence upon this subject is strongly opposed to such a view. However, until the nature and origin of pituitrin shall have been satisfactorily explained the matter must remain in doubt.

total extirpation is unjustifiable because of the extremely slender chance it has of accomplishing its object and the great hazard with which such an attempt must be associated.

Assuming, then, that the hypophyseal adenoma is to be considered incapable of complete extirpation, what can be done for the patient? If the neoplasm is not completely removed, it will certainly continue to grow (though its growth may be retarded by radiotherapy). Furthermore, it will probably continue to produce its noxious secretion if of the eosinophilic variety.

From surgery, therefore, we cannot as yet anticipate any permanent modification of the constitutional symptoms of acromegaly, even though experiences such as we have recorded (cf. *Case 6*) may point in this direction. Much, however, can be done to relieve the distressing consequences of local pressure of the tumour—the headaches and loss of vision; and we would emphasize that it is with this objective alone that the surgery of the hypophysis can be rightly employed.

We are dealing with a tumour which cannot be completely removed, which is enclosed by resistant structures below, and which consequently tends to grow upward and press upon the chiasm, thereby causing blindness. By removing the resistant structures below it, we can not only relieve the tension headaches, but also release the important structures above it from compression. This is one of the main objects of the transphenoidal operation,<sup>29</sup> which produces a decompression from below by removal of the sellar floor and splitting of the dural capsule. We would emphasize that the object of this surgical procedure is not only to remove as much of the tumour as thought safe, but also to relieve the upward pressure of a lesion which cannot be removed entirely, and will inevitably continue to grow, by permitting the growth to enlarge downward. Only the operation from below can accomplish this object. By approaching the tumour from above, it is possible in favourable cases to effect a partial removal and in this way to relieve temporarily the optic fibres from compression. But in the opinion of the Clinic the chief argument against this procedure lies in the fact that incision of the dural capsule, in those cases in which, though bulging, it is still intact, opens the way for a ready intracranial extension of the growth which might otherwise be long postponed.

Admitting the propriety of operating on pituitary adenomata from below, the very great importance of their correct diagnosis becomes evident, for it is particularly dangerous to operate on pituitary lesions other than adenomata by the transphenoidal route. Suprasellar tumours, and likewise the adenomata which have invaded the cranial chamber, must be approached from above, and in this Clinic an osteoplastic transfrontal exposure<sup>30</sup> is greatly preferred to that which approaches the lesion from the side. The transphenoidal operation, as a matter of experience, carries with it very little risk of meningeal infection when conducted in the presence of an adenoma with a ballooned sella.

In the series of 162 cases which have been subjected to operation the mortality has been: in 177 transphenoidal operations, 8.6 per cent; and in 19 transfrontal operations, 5.2 per cent. The transfrontal series is so small

that the figures are scarcely comparable. It is quite probable, nevertheless, that the transphenoidal operation will always have a slightly higher operative mortality than the transfrontal procedure, from the occasional possibility of meningeal infection which it carries with it. However, in dealing with a condition such as pituitary adenoma, an operative mortality of 8.6 per cent does not appear excessive. Even so, it may be said that these inclusive mortality figures are greatly affected by early experiences with advanced lesions and hopeless secondary operations. Since May 1, 1923, a consecutive series of 42 transphenoidal operations has been carried out with but one fatality.

A far more weighty consideration than the difference between an 8.6 per cent and a 5.2 per cent operative mortality is the degree of benefit which is conferred on the majority of the patients by each procedure. The transfrontal operation can hold out little to a patient with an adenoma beyond an exploratory operation with possible temporary relief of pressure symptoms; and there is always the risk of further damage to the chiasm by direct trauma, which defeats the main purpose of the operation. The transphenoidal procedure confers a large measure of relief whose duration is much longer, often extending over many years.

When, therefore, a diagnosis of adenoma can be made with confidence, we believe there is no question that the first surgical step should be a sellar decompression with partial removal of the tumour from below. Later, should definite signs of an intracranial extension persist, the advisability of an intracranial operation for its removal may have to be considered. This is especially to be contemplated when signs of a lateral intracranial extension are present, as evidenced by homonymous hemianopsia, uncinat attacks, etc. Such an extension is less likely to recede into the decompressed sella than is a median one. The transfrontal approach is the more satisfactory in such a case.

We do not consider that the doubtful gain in exposure which Heuer's<sup>31</sup> and Adson's frontolateral operation may offer compensates for the occasional cerebral injuries and much higher mortality-rate with which these procedures have been associated.

To summarize the treatment of hypophysial adenomata, we advocate: A trial of X-ray therapy unless there is imminent danger of loss of vision, when a sellar decompression should be done forthwith. During the course of X-ray treatment the progress of events should be carefully controlled by repeated perimetric examination. If visual impairment becomes greater, a transphenoidal operation should be carried out, with partial removal of the tumour. Later, should symptoms referable to a lateral intracranial extension of the tumour persist, a transfrontal operation should be undertaken. Following all these measures, X-ray treatments should be given in the hope of retarding the further growth of the tumour. They should at first be administered at intervals of three or four weeks, and later the treatments should be adjusted as the progress and circumstances of the particular case demand.

## SUMMARY.

1. A clinico-pathological study of 162 examples of pituitary adenomata shows that three fairly definite types of tumour can be histologically recognized and that they represent more or less definite clinical syndromes.

2. The syndrome of acromegaly corresponds with the chromophil adenoma, that of hypopituitarism with a chromophobe adenoma, and the mixed syndrome with a mixed type of adenoma. Case histories are given to illustrate these several syndromes.

3. Though our knowledge of the physiological action of the pars distalis is in its infancy, it may be asserted not only that it exerts an influence on growth, but that the eosinophilic cells are the ones chiefly concerned. In the absence of an increase in the number of these cells, evidences of overgrowth of one sort or another have not been found to occur.

4. We have as yet no knowledge of the biochemical influences which underlie the formation of these adenomata, and can only draw analogies from the better known lesions of the thyroid in arriving at our interpretations of them.

5. The treatment of these lesions is therefore still at the primitive stage in which a radical operative measure, rather than some simpler agent, is called upon to relieve the effects of local pressure on important neighbouring structures.

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**RIGHT DUODENAL HERNIA :  
WITH REPORT OF A CASE SUCCESSFULLY OPERATED ON,  
THE FOURTH RECORDED RECOVERY.**

BY FRANCIS R. BROWN, DUNDEE.

THE following is the report of a case of right duodenal hernia. The classification adopted is that given by Moynihan in 1899 in his monograph on retroperitoneal hernia. This very rare condition has also been described as right paraduodenal hernia, and as hernia of the mesentericoparietal fossa of Waldeyer.

The patient, a man of 49 years, was admitted to Dundee Royal Infirmary on Dec. 13, 1922, with the provisional diagnosis of chronic duodenal ulcer. Seven years before he fell from a height, and both bones of the right leg were fractured. At that time he thought he had injured something in his abdomen. He was off work for eighteen months, and 'had never felt the same since'. Three years before admission he underwent an operation for hæmorrhoids. He referred the onset of his present illness to an occasion about two months before admission. While at stool he was 'pressing pretty hard' owing to constipation, and thought he felt something 'go' in the region of the epigastrium. This was followed by pain of a steady, dull, aching character in the epigastrium for one month. He stated that his doctor told him there was a swelling there. (The doctor informed me that only on one occasion did he get the impression of a palpable swelling in the upper abdomen, and as he failed to verify it on subsequent examinations, did not attach any significance to the observation.) The pain, almost constant for a month, gradually became less severe, and latterly had practically disappeared. It had no time relationship to meals, and was neither aggravated nor relieved by taking food. The appetite was good, and there was no vomiting. On examination of the abdomen, nothing definite was made out except slight tenderness on pressure over and just below the xiphisternum. X-ray examination of the alimentary tract was reported to reveal no abnormality.

No definite diagnosis was made, and on Dec. 23, 1922, I performed laparotomy through a high right paramedian incision. On opening the peritoneal cavity no free small intestine was visible, but coils of small intestine were observed 'shining through' a thin layer of peritoneum, exactly as one often sees when dealing with a thin inguinal hernial sac containing unreduced bowel. The first impression received was that there was a second layer of anterior parietal peritoneum. Further investigation showed that the condition was an internal hernia into a large peritoneal sac. The mouth of the sac looked to the left; its posterior boundary was formed by the lumbar vertebræ covered by peritoneum, and in the anterior margin lay the superior mesenteric artery. The rest of the sac, occupying mainly the right half of the

abdomen, was freely movable, and the hand could be passed completely round between it (the sac) and the parietal peritoneum lining the posterior abdominal wall. The contents of the sac were rather more than the upper two-thirds of the small intestine, the jejunum entering the sac about three inches from the duodenojejunal junction, which appeared to be in its normal position. The herniated bowel was easily withdrawn, and was of normal appearance. The first part of the jejunum was free. After reduction of its contents, the greater part of the sac could be brought out of the abdomen,

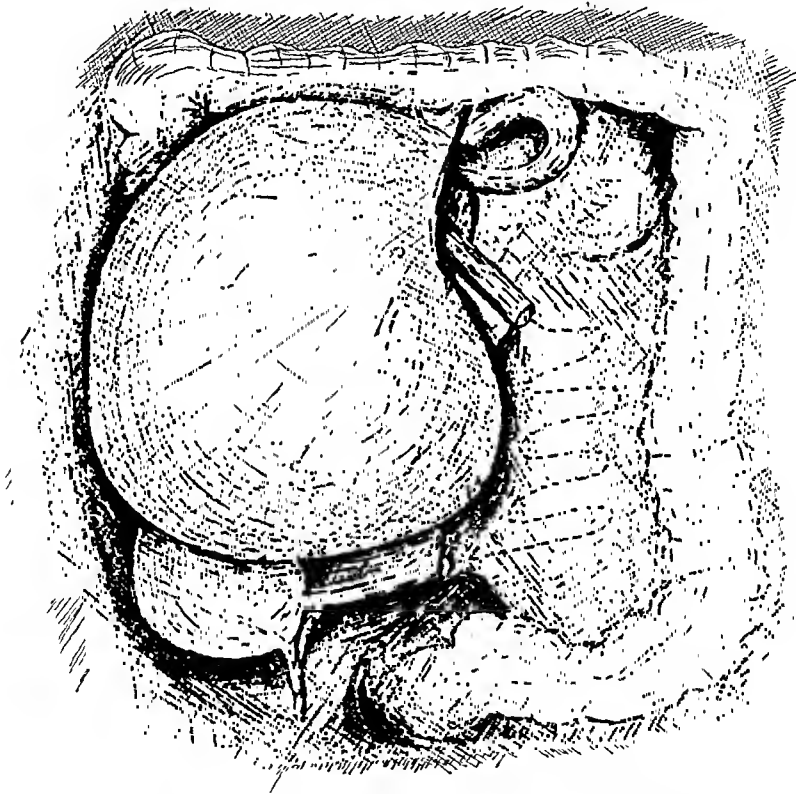


FIG. 250.—*Author's case.* Right duodenal hernia. The orifice of the sac is shown, with afferent jejunum and efferent ileum. Notice the superior mesenteric artery in the anterior margin. The fundus of the sac lies in front of the ascending colon.

and the situation of the orifice, which admitted four fingers, was verified. The sac was turned completely inside out so that the orifice looked to the right, the neck was closed by a catgut suture, and the fundus was removed. (*Figs. 250–252.*) The ascending colon had a definite mesentery, and the cæcum was unduly mobile, which enabled one to remove the appendix easily through the high incision. No other abnormality was observed. The patient made an uneventful recovery, and has had no return of his previous symptoms.

FIG. 251.—*Author's case.*  
Diagram of transverse section  
at level of mouth of the sac.  
Shows the sac lying in front of  
the ascending colon and meso-  
colon.

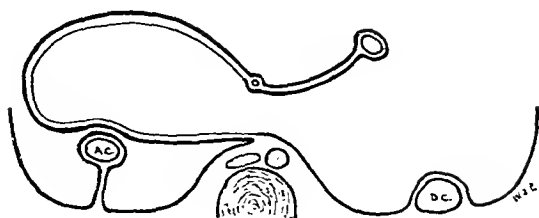


FIG. 252.—*Author's case.*  
Method of obliteration of sac  
by eversion and closure of the  
neck by suture.

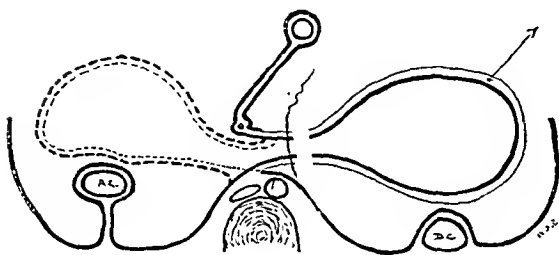


FIG. 253.—Diagram of  
transverse section to show the  
normal disposition of the meso-  
entery and peritoneum of the  
posterior abdominal wall—for  
comparison with *Figs.* 251,  
252, 254, and 255.

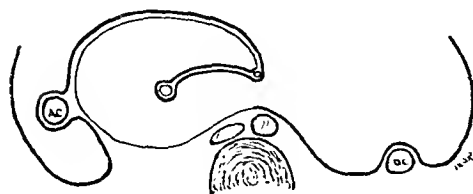
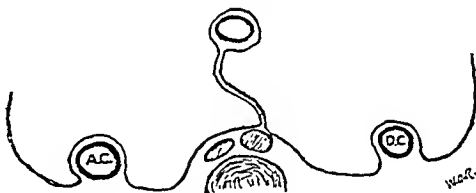
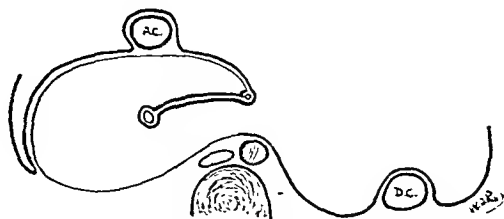


FIG. 254.\*—Sac passing  
behind the posterior parietal  
peritoneum. Ascending colon  
pushed outwards by the hernia  
when there is no ascending  
mesocolon.

FIG. 255.\*—Tho hernia has  
burrowed behind the ascending  
colon, and appears on its outer  
side. This is the condition  
commonly found when an as-  
cending mesocolon is present.



\* *Figs.* 254 and 255 are diagrams of transverse sections to illustrate the position of the sac in the more usual types of right duodenal hernia.

## DISCUSSION.

Moynihan<sup>1</sup> lays down three conditions as invariably present in right duodenal hernia : (1) The sac occupies—at any rate, at first—the right half of the abdominal cavity, lying behind the ascending and transverse mesocolon ; (2) The orifice is situated behind and to the left of the sac, on the lumbar vertebræ ; (3) In the anterior margin of the sac there lies either the superior mesenteric artery or a continuation of it, the ileocolic artery.

In the case here recorded, the position of the orifice, and the presence of the superior mesenteric artery in the anterior margin of the orifice, are in accord with Moynihan's criteria, but the position of the sac in relation to the posterior parietal peritoneum is unusual. Instead of stripping the posterior parietal peritoneum off the posterior abdominal wall, and burrowing behind the ascending colon and mesocolon, the sac lay freely in front of these structures, being indeed fixed only at its orifice. (*Figs. 251, 254, 255.*)

The presence of an ascending mesocolon has been noted in previously reported cases, e.g., by Broesike, by Moynihan, and by Carson.<sup>2</sup> Gruber<sup>3</sup> reported a case associated with a persistent mesentery for the large bowel, and attached considerable significance to this finding. Nagel,<sup>4</sup> in describing his case, states that "the presence of a persistent mesentery to the ascending colon may have aided in forcing the intestines into the pocket, and it undoubtedly helped to maintain them there".

Abnormalities about the duodenojejunal flexure appear also to be common in this condition. Thus Broesike,<sup>5</sup> in the cases he investigated, found a fusion of the upper few inches of jejunum to the posterior abdominal wall, and believed that this abnormality was essential for the formation of this type of hernia. Moynihan has shown that this condition is not necessary, and that the jejunum may have a free mesentery from its commencement—as in the author's case and others, e.g., Barrs',<sup>6</sup> and Griffith's museum specimen in Leeds Infirmary. On the other hand, the ascending portion of the duodenum may have a free mesentery continuous with the mesojejunum—as found in the cases described by Carson, and Bernardbeig.<sup>7</sup>

Much significance cannot, I think, be attached to the history of hæmorrhoids in this case, although it is conceivable that in some cases there may be pressure on the inferior mesenteric vein, which vessel, Carson states, was present in the posterior margin of the neck of the sac in his case ; this feature no doubt caused the enormous dilatation of the veins of the descending colon and sigmoid which he noted. The occurrence of hæmorrhoids in left duodenal hernia (from compression of the inferior mesenteric vein) has been reported (Strazewski<sup>8</sup>), and Moynihan refers to an article by Leichtenstern,<sup>9</sup> who first pointed out the diagnostic significance of this observation.

## ORIGIN OF RIGHT DUODENAL HERNIA.

Several somewhat conflicting theories have been brought forward to account for the formation of this type of internal hernia, but as yet it is doubtful if the true explanation is known.

Sir A. Cooper's case of 'mesenteric hernia'—almost certainly of the same nature as the author's—was supposed by him to result from traumatic rupture

of one layer of the mesentery, and the protrusion through the aperture of intestine which would force in front of it the other intact layer as a hernial sac, or from congenital defect in either layer, when a similar effect would ensue.

According to Broesike and Moynihan, right duodenal hernia originates only in the mesentericoparietal fossa, first described by Waldeyer in 1874. Moynihan's description is as follows: "The most usual position of this fossa is in the first part of the mesojejunum, immediately behind the superior mesenteric artery and immediately below the duodenum. The fossa varies considerably in size. The fossa has its orifice looking to the left, its blind extremity to the right and downward. In front it is bounded by the superior mesenteric artery, and behind by the lumbar vertebræ. The peritoneum of the left leaf of the mesentery lines the fossa; that of the right covers the blind end, and is then continued directly into the posterior parietal peritoneum. A forcible enlargement of the fossa would thus result in a tearing up of this layer of peritoneum lining the posterior abdominal wall".

Klob,<sup>10</sup> the first to describe this condition accurately, and later, Jonnesco,<sup>11</sup> believed the hernia to originate in the inferior duodenal fossa. More recently, Nagel writes: "While the majority of right duodenal hernias probably arise in the fossa of Waldeyer, it is reasonable to assume that some of them may originate in the inferior duodenal fossa when situated very low, near the beginning of the third portion of the duodenum and over the center of the vertebral column, or even to the right of the middle line". The usual position of the inferior fossa is high and to the left of the middle line. This low position was found in four of the bodies examined by Nagel. "Herniation into such a fossa", he writes, "would naturally progress toward the right, and by a peeling back of its superior peritoneal margin, until it reached the mesenteric vessels, would form a typical right paraduodenal hernia".

Edmund Andrews<sup>12</sup> advances an embryological explanation of the causation of duodenal hernia, which term he considers a misnomer. He believes that the condition is "a congenital anomaly due to imprisonment of the small intestine beneath the mesentery of the developing colon". He characterizes as absurd and grotesque the prevalent conception that these enormous hernias originate in small peritoneal pouches, and grow in size by gradual expansion of the sac. In support of his contention, he states: (1) Differential pressure within the abdomen is utterly lacking, so that any *vis a tergo* to account for the formation or growth of such hernia is totally absent. (2) There are literally hundreds of similar folds and fossæ in the peritoneum, many of which are of much greater size, and they are practically never the sites of such 'hernias'. (3) In all but a very small minority of cases reported, the degree of herniation has been total or subtotal. (4) A case of total hernia in a new born infant has been reported (left duodenal hernia—Vogt). Andrews writes "Surely one cannot believe that intra-abdominal pressure in utero has been the cause of such a 'hernia' ". (5) The herniated viscera are never anything but small bowel. The presence of omentum in the sac has never been reported. In only one case, that of Pybus (left duodenal hernia), was a few inches of the descending colon herniated. (6) In many of the cases there has been an almost universal growing together of the contents of the sac. This

condition, termed 'totalis accreta' by Kohlman, was present in Andrews' case, and he considers the adhesions to be derived from retroperitoneal tissue rather than the result of inflammation. Andrews explains production of right duodenal hernia as follows: "Suppose that the rotation of the umbilical loop is not carried to completion. The cæcum then would not lie superior to the small intestines" (Andrews illustrates it lying at a lower level on the left side of the abdomen), "and as it grew to the right, the small bowel would be caught in its mesentery, and finally, when its mesentery became adherent to the right posterior abdominal wall, its imprisonment would be complete. The superior mesenteric and ileocolic artery would lie in the free edge of the sac. All the conditions of a right duodenal hernia are exactly reproduced". He explains the formation of left duodenal hernia as "merely a higher degree of the same process", and believes that cases of mesocolic hernia and pericæcal hernia may have a similar origin.

Against Andrews' theory, at first sight a plausible explanation, can be made the following criticism. Normally, during the second stage of rotation of the mid-gut loop at the tenth week, the small intestines pass to the left side of the abdomen behind the superior mesenteric vessels; the cæcum then passing to the right side and eventually descending to the right loin as the colon elongates; the ultimate result of this rotation being an anti-clockwise turn of  $270^\circ$  about the axis of the superior mesenteric artery (Dott).<sup>13</sup> When this rotation does not occur, the small intestines occupy the right hypochondriac, lumbar, and iliac regions. The colon is confined to the left side. The small intestine, so situated, would not only effectively prevent the ascending colon and mesocolon from reaching and adhering to the right posterior abdominal wall, but, instead, would tend to perpetuate the left-sided position of the ascending colon, which would thus lie alongside the descending colon. Many cases of this type of non-rotation have been reported, the chief surgical interest being left-sided appendicitis.

In the author's case, the hernial sac lay, as already emphasized, freely in front of the ascending colon and mesocolon, a position which is inconsistent with Andrews' embryological explanation.

### SUMMARY OF RECORDED CASES OF RIGHT DUODENAL HERNIA.

Nagel collected and summarized 29 cases, including his own; of these, 17 were noted by Moynihan<sup>1</sup> in 1906. Rendle Short,<sup>14</sup> in his paper on retroperitoneal hernia, confines himself to those cases producing symptoms during life and in which an operation was performed. To Nagel's list of such cases, he added that of Novak and Sussmann,<sup>15</sup> a man, age 34, who recovered—making thus a total of 3 recoveries and 10 deaths. Two more authentic cases are now added to the list: Bernardbeig's, a man of 56, operated on unsuccessfully for acute obstruction in November, 1924; and the author's, operated on in December, 1922, and now recorded.

Levin and Ellis<sup>16</sup> reported a case of hernia into the inferior duodenal fossa in a boy of 8 years who recovered after operation. Levin considers it to be a right duodenal hernia. At operation about 15 to 18 in. of the commencement of the jejunum were found in a sac in the mesentery to the right

of the vertebral column. The mouth of the sac looked upwards and to the left, and was in the situation of the inferior duodenal fossa. The superior mesenteric artery was not identified, and Levin states: "In this particular case, I am quite certain that there was no vessel in the anterior boundary of the neck, for after I had made my incision over the 'tumour' I slipped my finger into the constricting ring and cut through it: there was no bleeding, therefore there could not have been a vessel of any size in the neck". This case appears to be similar to Molin's hernia into the inferior duodenal fossa, and cannot, I consider, be accepted as a right duodenal hernia.

*Table I (after Nagel).—RECORDED CASES OF RIGHT DUODENAL HERNIA.  
(AUTHENTIC CASES.)*

OBSERVER	YEAR	AGE	SEX	WHEN DIAGNOSED	SYMPTOMS OF ACUTE OBSTRUC- TION	OPERA- TION	RESULT
1 Klob .. ..	1861	36	M.	At necropsy	—	No	—
2 Gruber .. ..	1868	25	M.	At necropsy	—	No	—
3 Moutard-Martin ..	1870	—	—	At dissection	—	No	—
4 Guy's Museum specimen .. ..	{ 1870 1871	—	—	Museum specimen	No	No	—
5 Fürst .. ..	1884	61	M.	At necropsy	—	No	—
6 Zwaardenmaker ..	1884	Adult	M.	At necropsy	Yes	No	—
7 Quénu (Jonnesco)	1885	50	M.	At operation	Yes	Yes	Death
8 Gerard-Marchant..	1885	—	—	At dissection	—	No	—
9 Broesike .. ..	{ 1884 1885	2	M.	At dissection	—	No	—
10 Broesike .. ..	1886	Adult	M.	At dissection	—	No	—
11 Barrs .. ..	1891	18	M.	At necropsy	Yes	No	—
12 Clarke .. ..	1893	Adult	M.	At operation	Yes	Yes	Death
13 Rose .. ..	1895	68	F.	At operation	Yes	Yes	Death
14 Morestin .. ..	1896	Adult	M.	At dissection	Yes	No	—
15 Neumann .. ..	1898	55	F.	At operation	Yes	Yes	Cure
16 Griffith (Moynihan)	1898	—	—	Museum specimen	—	No	—
17 Koppers .. ..	1899	—	—	—	—	—	—
18 Schwalbe .. ..	1903	14 months	M.	At dissection	—	No	—
19 Schwalbe .. ..	1903	Adult	M.	At dissection	—	No	—
20 Selby .. ..	1904	40	M.	At operation	Yes	Yes	Death
21 Patou .. ..	1906	3 months	—	At operation	Yes	Yes	Death
22 Haasler .. ..	1907	43	F.	At necropsy	—	Yes	Death
23 Haasler .. ..	1907	10	M.	At operation	Yes	Yes	Death
24 MacCullum and Miller .. ..	1908	41	M.	At operation	Yes	Yes	Death
25 Van Rossum .. ..	1909	—	—	—	—	—	—
26 Merrigan .. ..	1911	—	—	At dissection	—	—	—
27 Mueller .. ..	1911	47	M.	At operation	Yes	Yes	Death
28 Carson .. ..	1912	29	M.	At operation	Yes	Yes	Cure
29 Nagel .. ..	1923	55	M.	At necropsy	No	Yes	Death
30 Novak and Siessmann .. ..	1924	34	M.	At operation	Yes	Yes	Cure
31 Bernardbeig .. ..	1925	56	M.	At operation	Yes	Yes	Death
32 Brown .. ..	1925	49	M.	At operation	Chronic	Yes	Cure

*Table I* is a summary of the 32 authentic cases, including my own, in the literature. Of these, 15 were operated on; 15 had symptoms of acute

obstruction, and 3 of these died without operation having been performed. In 2 cases (Nagel's and one of Haasler's), although laparotomy had been performed for other conditions, the presence of right duodenal hernia, found at necropsy, was unsuspected during life, and it apparently had given rise to no symptoms. Thus 13 cases were operated on because of symptoms produced by the hernia, and there were 4 recoveries.

### CLINICAL FEATURES AND DIAGNOSIS.

So far, not a single case of right duodenal hernia has been diagnosed before operation or necropsy.

It may be observed at any age. The youngest patient was that of Paton, age 3 months; the oldest, reported by Rose, was a woman of 68 years. In the male sex 21 cases occurred, and 3 in females; in 8 cases the sex is not noted.

The great majority were operated on for symptoms of acute obstruction, which may follow several similar attacks or suddenly occur in an apparently previously healthy patient. In the more chronic type the symptoms have been likened to those of chronic duodenal ulcer, but they resemble rather those of chronic duodenal ileus. In others there may be more or less severe persistent pain with little or no vomiting, as in chronic obstruction. Lastly, the condition may remain symptomless, and be found at necropsy. The most important physical signs are:—

1. *Vomiting*.—This may be absent or scanty even when obstruction is complete. If continued, the vomitus will consist mainly of bile: there can be no regurgitation of small intestine contents. In Carson's case constant vomiting of bright-green material was a feature.

2. *Visible Peristalsis*.—A sign of chronic obstruction occurring in the bowel above the site of constriction. In Carson's case, and in that of Levin and Ellis, peristaltic waves passing from left to right across the epigastrium were observed. In both these cases, subsequent operation showed dilatation of the stomach and duodenum. While marked peristaltic waves of the herniated intestine may occur if the efferent loop is obstructed, it is doubtful if such peristalsis would be visible except in a very thin patient, owing to the deep position of the hernia behind the ascending colon and mesocolon—the most common situation.

3. *Palpable Swelling*.—This, the most important diagnostic sign when present, is described by Moynihan as “a palpable, definite, resonant mass which lies at first to the right and lower part of the abdomen, but spreads finally over almost the whole abdominal cavity.” The tumour may bear a very obvious relation to the clinical condition of the patient, becoming more tense and prominent and very much more tender when the symptoms undergo exacerbation. As the symptoms decline in severity, the tumour becomes less aggressive. On auscultation, distinct, gurgling sounds may be heard anywhere in the tumour”. In Carson's case the percussion note over the tumour was dull, and at operation the small intestine was withdrawn in a collapsed condition from the sac, which of course would explain the lack of resonance on percussion.



4. *Result of X-ray Examination.*—(a) Where there is no obstruction. In Nagel's case X-ray examination carried out two months before death showed the colon filling the left half of the abdomen, with coils of small intestine grouped to the right. This appearance was considered to represent incomplete intestinal rotation, and no further significance was attached to it. (b) Where there is chronic obstruction of the afferent loop, the X-ray picture will be similar to that found in chronic duodenal ileus—namely, dilatation of the stomach and duodenum with delay in emptying.

5. *Toxaemia and Collapse.*—These will be intense in acute obstruction if and when practically the whole of the small intestine is strangulated.

### TREATMENT.

As long ago as 1906, Moynihan wrote: "In a certain proportion of cases it is likely in the future that an operation will be undertaken after a correct diagnosis has been made". While in a few cases of left duodenal hernia the condition was clinically diagnosed, there is still, as already mentioned, no similar record amongst those of the right variety.

Obstruction, when present, may be due to: (1) Constriction of the intestine by the orifice, although in most cases this is fortunately so large that strangulation does not occur. (2) Adhesions about the orifice of the sac. Thus Bernardbeig states that in his case the orifice was reduced in size by adhesions, which were broken down during the operation, and which played a very important part in the strangulation. (3) Axial twisting or volvulus of the intestine at the neck of the sac, as reported by several writers (Neumann, Carson, Bernardbeig, and others). The author suggests that obstruction may also occur outside the sac, and affect the bowel proximal to the orifice. The weight of the herniated intestine in its dependent sac will so drag on the root of the mesentery that constriction of the third part of the duodenum by the cord-like superior mesenteric artery is likely to result. This would give rise to the condition of duodenal ileus. In such an event, there would be no sudden increase in size of the 'tumour', since the efferent loop is not necessarily constricted.

Reduction, by traction on the afferent or efferent loop of intestine, is generally easy, and spontaneously relieves the obstruction, however caused. Should the orifice have to be enlarged, wounding of the superior mesenteric artery must be carefully avoided. The inferior margin only of the orifice may be incised.

If distention of the strangulated gut prevents reduction, the sac should be entered through an avascular space to the right of the superior mesenteric artery, and the bowel withdrawn, opened, and emptied, after which reduction through the orifice should be easily accomplished.

After reduction, closure of the neck of the sac should be attempted, if the condition of the patient permits, in order to prevent the possibility of recurrence. In at least two cases (Paton's and Mueller's), where this procedure was not carried out, it was found at post-mortem examination that a considerable quantity of gut had re-entered the sacs.

Partial obliteration of the neck of the sac by suture was performed in

two of the previously reported successful cases (Neumann, and Novak and Sussmann). In Carson's no attempt was made to close the opening. In the author's case the disposition of the sac rendered eversion, and closure of the neck, a comparatively simple procedure.

For the figures illustrating this article, I am indebted to Mr. W. O. Reid, Dundee.

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## SOME UNUSUAL MANIFESTATIONS OF SPREAD BY IMPLANTATION OF PAPILLOMATA OF THE URINARY TRACT.

BY B. C. MAYBURY, LONDON, AND S. C. DYKE, WOLVERHAMPTON.

FEW types of new growth exemplify better the process of spread by direct implantation than the papillomata of the urinary tract. The facility with which, by the spontaneous dissemination of seedlets, those of the bladder spread over the mucous membrane of that organ is one of their best recognized characteristics, and their recurrence as the result of accidental implantation in operation wounds in the vesical wall is not rare. The two cases described below are reported with the view of drawing attention to some less common manifestations of this power of spread by implantation. A further point of very great interest in both cases is the rapid growth of secondary tumours after the removal of the primary ones. In view of the fact recently demonstrated by Murray,<sup>1</sup> that the presence of a primary cancerous tumour exercises an inhibitory effect upon the growth of implants of tumours of a similar type, it would appear that something more than coincidence was concerned in this phenomenon. In the first case the original tumour, though histologically of exactly the same type as a vesical papilloma, occurred in the renal pelvis, upon the epithelial lining of which structure implantation and growth of the seedlets occurred. In the second case implantation and growth of seedlets from a vesical papilloma took place altogether outside the urinary tract in the tissues of the abdominal wall; the recurrences, three in number, were remarkable in that, while the first, like the primary growth, was histologically simple, the succeeding recurrences showed a progressive change towards malignancy.

The first case was that of a male, age 45, with a history of repeated attacks of hæmaturia extending over a period of six years. As a rule the bleeding was unaccompanied by pain, but severe left-sided renal colic occurred during the first attack. One bout of hæmaturia, which lasted a month, followed on the patient's being thrown out of a car; but with this single exception he could assign no cause for the attacks. For the last twelve months the hæmaturia had been constant, the patient never having passed a clear specimen. The urine had been examined for organisms more than once, but cultures had invariably proved sterile, and no tubercle bacilli had been found.

When first seen the patient was somewhat thin, but not very anæmic, in spite of the prolonged and continuous loss of blood. Clinical examination revealed nothing abnormal; neither kidney could be palpated. Skiagrams showed no abnormal shadows. The urine was of a port-wine colour, acid, and

contained protein ; microscopically there were many epithelial cells and large numbers of red and pus cells, the two latter being present in approximately equal numbers ; no casts were seen ; cultures were sterile and no tubercle bacilli were found. Cystoscopic examination under an anæsthetic showed that the blood was coming from the left ureter, the jets from the right being normal and becoming coloured with indigocarmine in seven minutes. The bladder itself was normal. A diagnosis of papilloma of the pelvis of the kidney was made, and, after a second cystoscopic examination to confirm the previous

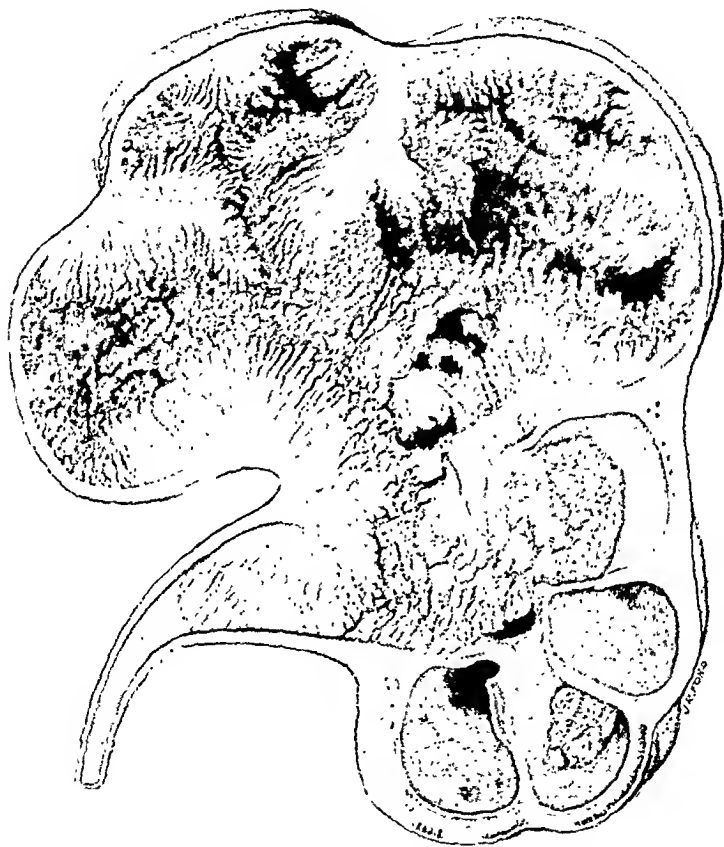


FIG. 256.—Mesial section of kidney removed from Case 1.

findings, nephrectomy was performed by the loin route on the left side. The patient made an uninterrupted recovery from the operation.

The kidney, half of which is shown in *Fig. 256*, is considerably enlarged. Growing from the pelvis, and partly obstructing the lumen, is a large papilloma which has caused dilatation of the calices. Secondary seedlets of smaller size and presumably younger can be seen in other parts of the hydronephrotic sac, and a group quite separate from the primary tumour is present in one of the lower calices.

Examination, both of the large papilloma at the outlet of the pelvis and also of other smaller ones taken at random from elsewhere in the wall of the hydronephrotic sac, shows them all to be of exactly the same type. They consist of a filiform and branching connective-tissue core containing well-formed blood-vessels, covered with many layers of epithelium of transitional type (*Fig. 257*). The histological appearances are exactly similar to those of an ordinary papilloma of the bladder. The epithelial cells, both in their individual appearance and their general arrangement, conform closely to the

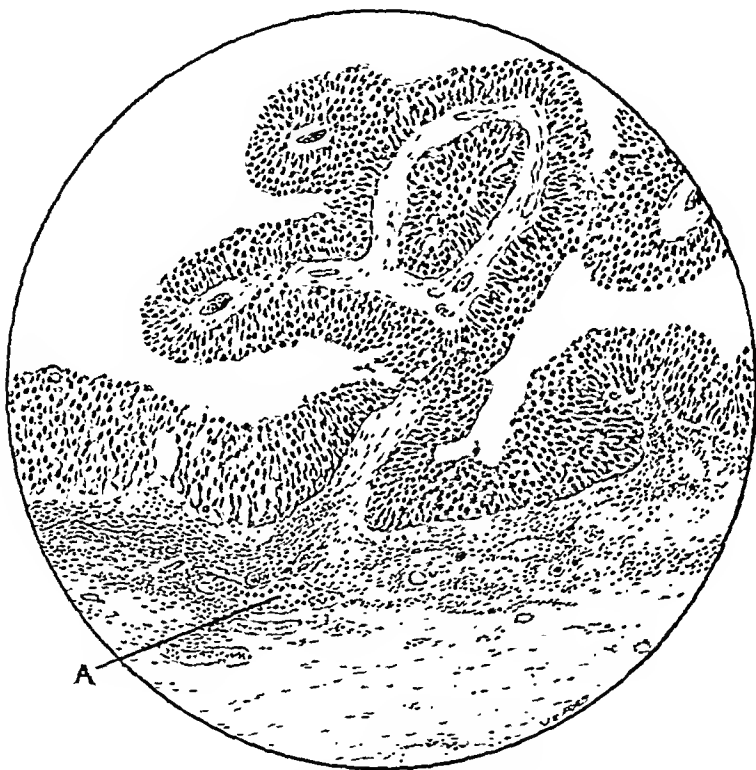


FIG. 257.—Microscopical appearance of the papilloma of the pelvis of the kidney shown in *Fig. 256*. The tumour is similar to an ordinary papilloma of the bladder. There is no evidence of invasion by the epithelial cells. Note the polymorphs present in the vessel at A.

transitional type. A portion of the pelvic wall was included in each piece of tissue sectioned, but in no instance was any indication of invasion by the epithelial growth to be seen. On this evidence, and on the typical nature of the tumours, the papillomata were deemed, in spite of their spread by dissemination, to be benign.

The pelvic wall was greatly thickened by fibrosis, and showed considerable infiltration by inflammatory cells of the plasma-cell type beneath the mucous membrane. No polymorphs were present in these collections of inflammatory

cells, but many of the smaller vessels in the subepithelial tissue were filled with them. The pelvic epithelium between the papillomatous growths was thickened, and consisted of more layers than normal.

The presence of pus cells in the urine in such large numbers as to cast some doubt upon the provisional diagnosis is a point of some interest in this case. Their occurrence in infinitely greater numbers than could be accounted for by the amount of blood present suggested the presence of an infective condition in the kidney, which, however, the results of culture of the urine and subsequent microscopical examination of the kidney proved not to be the case. In the description of the histological appearances of the tumour given above, it was pointed out that signs of a mild and chronic inflammatory process were present in the pelvic wall, but that polymorphs were not among the infiltrating cells. Attention was, however, drawn to the fact that such cells had collected in large numbers in the smaller vessels; a vessel containing such polymorphs is shown at A in *Fig. 257*. This stagnation of the polymorphs in the smaller vessels has long been recognized as one of the earliest manifestations of an inflammatory process and as preceding their actual passage by diapedesis from the vessels. It is the blood from the smaller superficial vessels which passes into the urine in cases of papillomata of the urinary tract, and the stagnation of the polymorphs in these vessels would seem to account for their excess in the blood thus passed.

Nine months after the operation, the patient returned with a month's history of recurrence of hæmaturia of exactly the same type as previously experienced. He looked ill and had lost a considerable amount of weight. On examination, the right kidney was found to be enlarged and hard but movable. A provisional diagnosis of papilloma of the remaining kidney was made, but in view of the serious condition of the patient and the impossibility of further operative treatment no cystoscopic examination was undertaken.

The renal swelling increased rapidly in size and later became fixed; from it there extended a large, hard, finely nodular plaque, which was adherent to the anterior and lateral abdominal wall and finally reached to well below the level of the anterior superior iliac spine. The patient became more and more emaciated, and died three months after the recurrence of the hæmaturia. Throughout there was neither any sign of local recurrence on the left side, nor apart from the extreme emaciation was there any clinical evidence of metastases.

Unfortunately there was no autopsy, but there can be little doubt but that the tumour on the right side arose in the kidney and was malignant in nature; probably it commenced as a papilloma similar to the growth removed with the left kidney.

It is conceivable that the removal of the papilloma with the left kidney had the effect of withdrawing the inhibitory effect of this primary tumour upon an existing similar tumour in the right kidney and thus enabled the latter to grow and later become malignant in nature.

In the second case, the patient, a female, 57 years of age, was admitted to St. Thomas's Hospital on Oct. 1, 1923, with a six months' history of attacks of painless hæmaturia. Physical examination was negative. The

urine was acid and contained red and pus cells. Cystoscopic examination demonstrated a moderate-sized, pedunculated papilloma free from phosphatic deposit growing from the trigone below and obscuring the right ureteric orifice. On Nov. 7 the tumour was removed through a long suprapubic incision. It was found to be of the ordinary villous type, about the size of an almond, and attached by a fairly narrow pedicle to the vesical wall, a portion of which was excised with it. The resulting raw area was obliterated by approximating the cut edges of the vesical wall with catgut sutures. The suprapubic wound was closed, and a drain inserted down to the suture line in the bladder. A urinary fistula formed, but healed in a month. On Jan. 1, 1924, cystoscopy showed the vesical scars quite sound and no sign of recurrence. The patient was discharged a few days later.

The specimen as submitted for microscopy consisted of a small portion of the bladder wall about 1.5 cm. long, on which was growing a villous papilloma. Microscopically the tumour showed the ordinary transitional type of epithelium; mitoses were not particularly in evidence, and both the appearance and general arrangement of the cells were quite typical. The base of the papilloma showed no tendency to invade the wall of the bladder, and it was passed without hesitation as benign, so far as morphological criteria avail in making a decision.

On May 7, 1924, six months after the original operation, the patient was readmitted complaining of a gradually increasing painful swelling of three months' duration in the suprapubic scar. She had had no recurrence of

hæmaturia. On examination there was a fairly well-defined, firm, oval, tender tumour about the size of a hen's egg, situated in the abdominal wall immediately beneath and adherent to the upper part of the suprapubic scar. The approximate site of the tumour is shown in *Fig. 258, A*. The skin overlying it was inflamed, and fluctuation was present in the centre. On the supposition that the swelling was an abscess, it was incised on May 10, but only blood-stained fluid and some friable 'granulation tissue' were evacuated. On May 19 the tumour was widely excised, portions of both rectus muscles and their sheaths being removed with it; the peritoneal cavity was not opened. Some difficulty was experienced in closing the wide gap in the abdominal wall. The bladder was examined with the cystoscope and was found free from recurrence. The patient made an uninterrupted recovery, and was discharged on June 23.

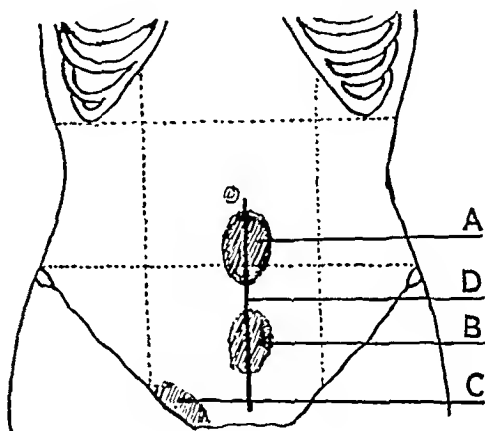


FIG. 258.—Diagram showing the approximate sites of 'graft-recurrences' in the abdominal wall after the suprapubic removal of a simple vesical papilloma (*Case 2*). A indicates the site of the first, B the second, and C the third recurrence, respectively. The suprapubic scar of the original operation is indicated at D.

The tumour is shown in cross-section in *Fig. 259*. At the top of the specimen can be seen the skin of the abdominal wall with the upper part of the healed scar. The tumour itself is cut across, and is represented by a ring of white in the centre of which is a red mass of blood-clot. A very well-marked layer of fibrous tissue sharply limits its periphery from the surrounding structures. A portion of the specimen, including both the central tumour mass and the surrounding fibrous tissue, was removed for microscopy. The histological appearances are shown in *Fig. 260*. The central mass consists of epithelial cells of transitional type, together with a certain amount of fibrous stroma. The relation of these two elements to each other shows quite clearly that the stroma represents the central connective-tissue core of the papilloma upon which the epithelial cells are applied. In appearance and arrangement the epithelial cells conform absolutely to type; their only departure from the

strict normal is in the presence of numerous mitoses. As shown in *Fig. 260*, the margin of the epithelial cell masses is for the most part sharply defined where it meets the surrounding reactionary fibrous tissue. In one or two places, indeed, there does appear a tendency for processes of the epithelial cells to pass out into this fibrous barrier, which shows a fairly intense infiltration by mononuclear inflammatory cells.

These last facts, together with the frequency of mitosis noted above, were regarded as suspicious; but on consideration of the very typical nature of the tumour as a whole and its wide removal,



**FIG. 259.**—Half of the first tumour removed from the abdominal wall in *Case 2*. The skin, together with a portion of the healed scar, is seen at the top of the specimen. The cross-section shows the tumour as a ring of white, and in the centre a mass of red blood-clot. A very well-marked layer of fibrous tissue sharply limits the periphery. A portion of the left rectus muscle is seen at the bottom right-hand corner.

a favourable prognosis was given so far as recurrence was concerned.

On Aug. 13 the patient was re-admitted with a history of the recurrence of a small painful tumour in the lower part of the suprapubic scar shortly after she had left hospital; this had gradually increased in size and finally broken through the skin, since when it had continuously discharged a blood-stained fluid. On examination there was a swelling similar to the previous recurrence occupying a corresponding position deep to the lowest part of the suprapubic scar (*Fig. 258, B*); it was, however, somewhat smaller, rather firmer, and slightly less defined; there was no fluctuation. In the centre of the tumour there was a sinus surrounded by a prominent mass of reddish-blue granulation tissue partly covered by a thin layer of epithelium. From the sinus was discharged a certain amount of blood-stained fluid. On Aug. 16 the tumour was freely excised with about three-quarters of an inch of the surrounding healthy tissue; small portions of the parietal peritoneum and



bladder were included; the latter appeared normal, and was not attached to the tumour. The opportunity was taken of inspecting the inside of the bladder, and no sign of recurrence was seen. The cut edges of the various layers of the abdominal wall could not be approximated completely, but the wound was closed by suturing the skin under considerable tension.

The tumour is shown in cross-section in *Fig. 261* and is somewhat similar to the previous specimen. The most striking difference, however, is the absence of any definition of the periphery of the growth, it being impossible

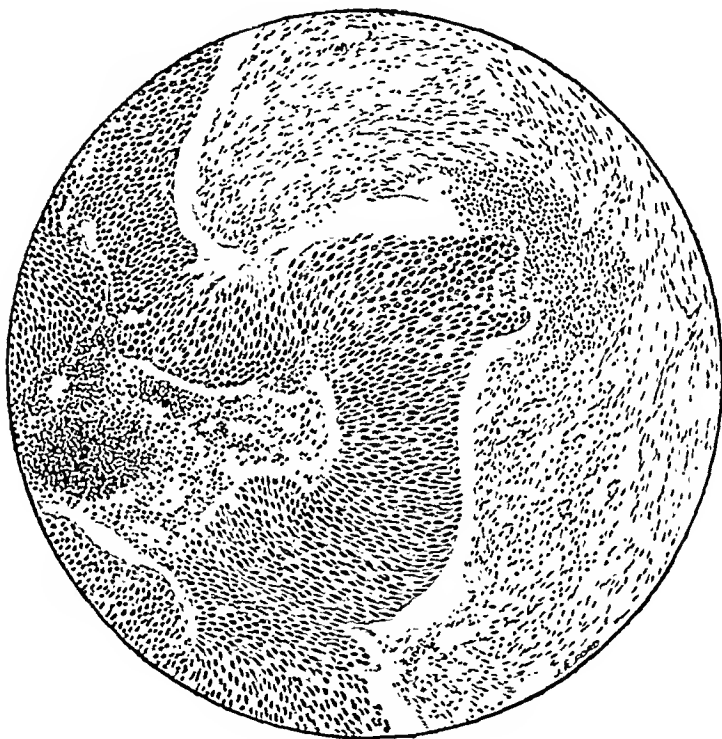


FIG. 260.—Microscopical section of the tumour shown in *Fig. 259*. The epithelial cells, of uniform size and shape, are arranged in masses upon a connective-tissue core. There is no definite invasion, although in one or two places there does appear a tendency for the epithelial processes to pass into the surrounding barrier of fibrous tissue.

in many places to see where the growth ends and the normal tissues begin. The extension of the tumour can be traced to the skin, where it forms a small cauliflower excrescence.

Microscopical examination showed this second recurrence to consist of transitional epithelial cells of the same type as seen in the first recurrence, but now of undoubted malignancy. As shown in *Fig. 262*, the typical arrangement around a central connective-tissue core has been lost, and the cells are disposed irregularly in relation to each other; the characteristic size and

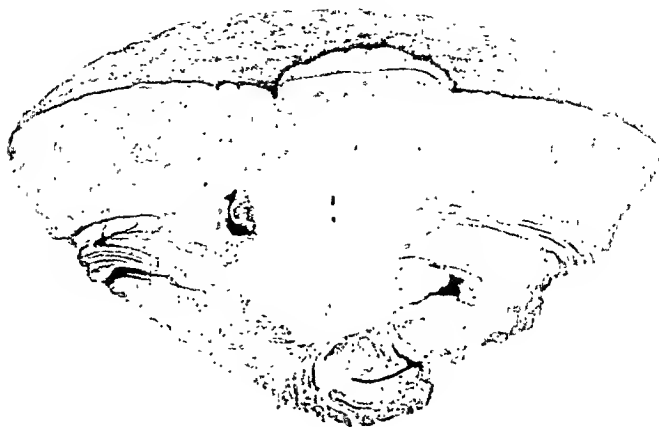


FIG. 261.—Half of the second tumour removed from the abdominal wall in *Case 2*. Contrast with *Fig. 259*. Note the absence of any definition of the periphery of the growth. The extension of the tumour can be traced to the skin, where it forms a small excrescence.

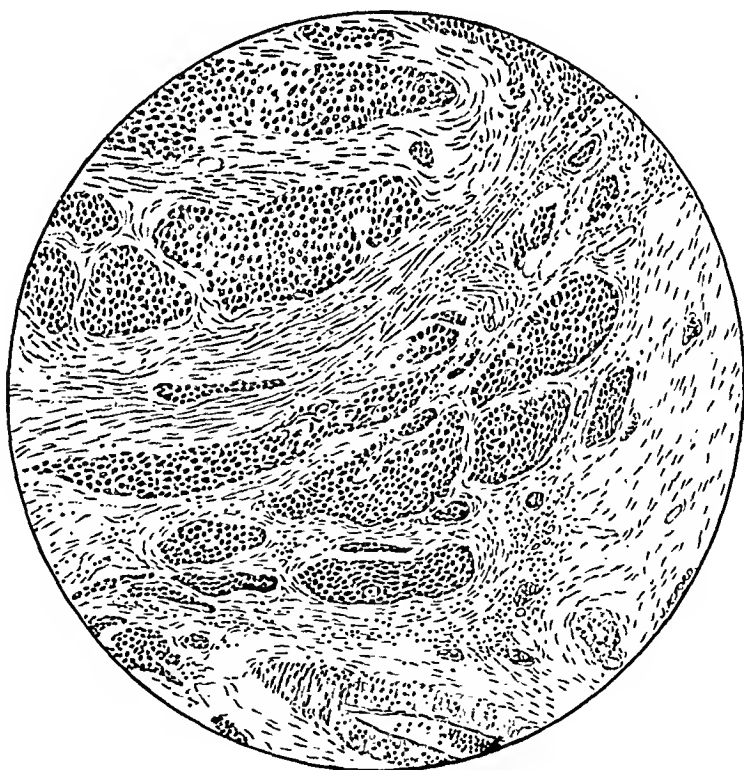


FIG. 262.—Microscopical section of the tumour shown in *Fig. 261*. Contrast with *Fig. 260*. The epithelial cells, of different sizes and shapes, are arranged in irregular masses, which are invading the surrounding fibrous tissue.

shape of the cells has largely disappeared. Finally, as shown in the illustration, the epithelial masses are clearly invading the surrounding fibrous tissue. Mitotic figures are of frequent occurrence in the epithelial cells; curiously enough, however, although all the other features, both histological and clinical, point to this recurrence as being of a much more malignant type than the preceding one, the actual number of dividing cells present in an equal number of fields is considerably less than in the first recurrence.

On Sept. 11, while the patient was still in hospital, a third recurrence was noticed deep to the muscles of the abdominal wall, firmly adherent to the inner portion of the right horizontal ramus of the pubes (*Fig. 258, c*). In view of the histological findings of the last tumour removed, and the fixation of the present one, further operative interference was considered useless.

The first recurrence of this tumour in the abdominal wound undoubtedly occurred as the result of implantation of seedlets from the original papilloma into the operation wound in the course of its removal. The fact that all three recurrences appeared at quite different sites in the abdominal wound, and the wide removal of the first two, preclude the possibility of the later recurrences having arisen from portions of their predecessors left behind at operation. There can be little doubt that all three recurrences arose from seedlets sown into the wound at the original operation.

### SUMMARY.

Two cases illustrating unusual manifestations of the power of spread by implantation possessed by papillomata of the urinary tract are reported.

In the first case the original papilloma occurred in the renal pelvis, and implantation took place on the walls of the resulting hydronephrosis. Removal of the affected kidney together with the tumour was followed by the rapid growth of a malignant tumour in the remaining kidney. The urine in this case showed much pus in the absence of any infective process; an explanation is offered of this phenomenon.

In the second case, after removal of a simple vesical papilloma, three 'graft-recurrences' occurred in three separate places in the tissues of the abdominal wall, each appearing shortly after the removal of the previous one; it is suggested that all three arose from portions of the original tumour accidentally implanted at the first operation. The primary tumour was histologically non-malignant, but the successive recurrences showed a steady progress towards malignancy.

It is suggested that the course of these cases offers an illustration of the inhibitory effect exercised by the presence of a primary tumour in the tissues upon the rate of growth of secondaries and their degree of malignancy.

Our thanks are due to Sir Cuthbert Wallace for permission to publish the notes of the second case.

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### REFERENCE.

<sup>1</sup> MURRAY, J. A., *Eighth Scientific Report of the Imperial Cancer Research Fund*, 1923.

*SHORT NOTES OF  
RARE OR OBSCURE CASES*

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**STRANGULATED FEMORAL HERNIA ASSOCIATED WITH  
AN APPENDIX ABSCESS IN THE HERNIAL SAC.**

By NORMAN HODGSON, NEWCASTLE-UPON-TYNE.

THE following case is of interest on account of the rare association of the two conditions and of the difficulty in diagnosis.

The patient, a woman, age 70, was admitted to hospital with intestinal obstruction of four days' duration. There was definite visible peristalsis of the small intestine. In the right femoral region there was a swelling the size of a walnut, with an active sinus on its inner side. There were no other physical signs. The patient stated that following an attack of abdominal pain eight months previously, this swelling appeared and burst, discharging pus; then it healed rapidly. She had had several mild attacks of abdominal pain, also recurrence of the abscess, but had not associated the two conditions. Since the first attack she had never felt well and had for the most part been confined to bed.

The diagnosis made was intestinal obstruction, probably due to a carcinoma of the colon, the swelling in the groin being thought to be a suppurating lymphatic gland.

Under general anaesthesia an incision was made into the swelling and a small quantity of pus evacuated. The abdomen was then explored through a median subumbilical incision. Collapsed small intestine presented, and following this down, it led into the femoral canal on the right side. Gentle traction delivered a knuckle of strangulated small intestine which quickly recovered its normal appearance. On inspecting the femoral ring, the appendix was seen running into the canal. It was firmly adherent to the posterior wall of the canal and was separated with difficulty. The appendix was removed and the stump buried. The abdomen was closed without drainage. The incision in the groin, which communicated with the hernial sac, was packed with gauze. The patient made an uneventful recovery.

Examination of the appendix showed a small perforation at its tip. The conclusion arrived at after operation was that the appendix lying in the hernial sac had become inflamed, perforated, and the resulting abscess had burst externally. The sac had remained unobliterated, and in it had occurred the strangulation for which the patient was admitted to hospital.

## TWO CASES OF HERNIA THROUGH THE TRANSVERSE MESOCOLON.

BY F. STRONG HEANEY AND G. C. E. SIMPSON, LIVERPOOL.

RENDLE SHORT,<sup>1</sup> in a recent article, summarizes sixteen cases of hernia through the transverse mesocolon. The writers call attention to the following three published cases which are not included by him.

Pringle<sup>2</sup> describes the case of a girl, 5 years old, which is certainly worthy of inclusion: here the opening must have been congenital.

Coffey<sup>3</sup> relates the case of a woman with a twenty years' history, who ten years before the operation had a sudden abdominal pain when carrying a heavy load of wood, following which she had been in bed for some months and had since been a chronic invalid. In this case the intestines traversed the lesser sac and emerged in part above the lesser curvature of the stomach.

P. T. Crymble<sup>4</sup> illustrates a case which he considers might have been a hernia through the transverse mesocolon. The hernia had emerged from the lesser sac through the gastrocolic omentum, but, like Pringle's fourth case, secondary adhesions were so marked that the exact relations were uncertain.

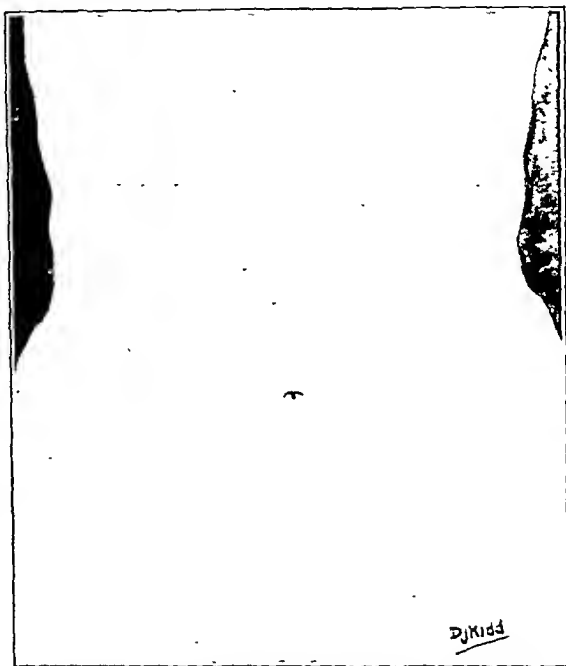


FIG. 263.—Case 1. Hernia through transverse mesocolon. External appearance.

The following are two hitherto unpublished cases:—

*Case 1* (G. C. E. S.).—A woman, 52 years of age, admitted to the Northern Hospital, Liverpool, in December, 1921, from a tuberculosis hospital. She had suffered from her stomach since girlhood. Twelve months previously she had had sudden *acute pain* in the stomach, and began to suffer more markedly from indigestion. Occasionally she had vomited, and at night brought back some food with a sour and bitter taste.

**EXAMINATION.**—She was extremely emaciated, and her abdomen showed two tumours, one in the upper part of the left abdomen and the other in the right abdomen about the umbilical level (*Fig. 263*). In these peristalsis

was visible from above and the left downwards to the right. Coils of small intestine were apparently overlapping the interval between the sacs.

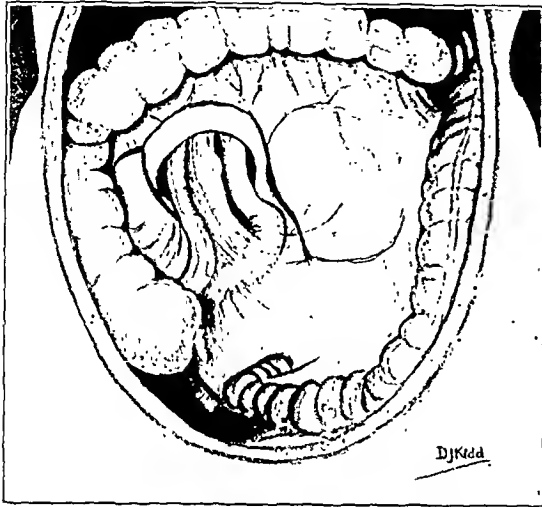


FIG. 264.—Case 1. View from below.

OPERATION.—In view of her extreme emaciation and the apparent certainty of the diagnosis of hour-glass stomach X-ray investigations were omitted, and the abdomen was explored; the two tumours were the halves of an hour-glass stomach. Gastro-enterostomy in the upper sac was undertaken in hopes that when the patient was in better condition it would be possible to resect the lower sac. On turning up the colon with a view to carrying out the usual posterior gastro-enterostomy, it was found

that there was practically no small intestine in the normal place (Fig. 264).

The terminal part of the ileum was soon identified, running a straight course downwards to the ileocaecal angle from a hole in the transverse mesocolon, while at the duodenojejunal junction the intestine continued its vertical course through the mesocolon. Traversing the lesser peritoneal sac, the small intestine emerged again above the stomach and cascaded down to the normal position (Fig. 265); both stomach and colon were greatly prolapsed. The intestine was readily withdrawn and brought to the normal position; a gastro-enterostomy was performed, and the hole in the transverse mesocolon sutured.

The patient made a good recovery, and was discharged on Jan. 4. She was re-admitted on March 15 with a view to resection of the pyloric sac,

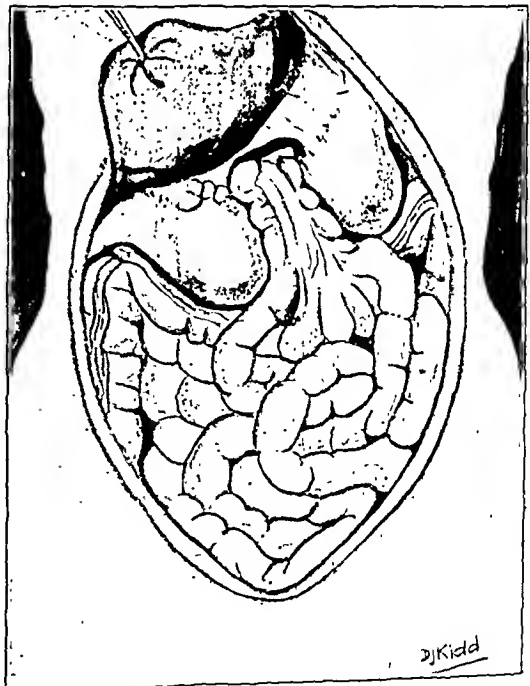


FIG. 265.—Case 1. Showing small intestine emerging from gastrohepatic omentum.

but the patient's doctor informed me that before sending her to hospital he had detected tubercle bacilli in the sputum. This observation was confirmed, and skiagrams of her chest lent further support to the diagnosis. Her heart also showed disease, and operative treatment was reluctantly abandoned.

The patient died of heart failure in November, 1922, and by the kindness of Dr. Macwilliam, of Walton Institution, Liverpool, I was present at the post-mortem. The patient had certainly added weight and was much better nourished, and the small intestines were in the normal place. The stomach was still in hour-glass form, the aperture between the two sacs being only about the diameter of a finger. On opening the stomach no signs of an ulcer or the scarring of an old ulcer were detected, although there was duodenal ulceration.

The cause of death was marked 'heart disease'. The specimen is now in the Museum of the University of Liverpool.

*Case 2 (F. S. H.).*—Mrs. P., age 38, first seen in August, 1923, gave a history of digestive trouble for sixteen years. Eighteen months previously she had a large gastric hæmorrhage and was confined to bed for five weeks. Subsequently she had to remain in bed from time to time for relief of gastric and general abdominal pain. For the last six weeks she had been vomiting after food, vomiting when she tried to walk, and in pain at all times save when lying down.

For years, concurrently with her other symptoms, she had suffered from constipation. She had tried to wear a visceroptosis belt, but it aggravated her epigastric pain.

**EXAMINATION.**—She was very emaciated, had a long thorax, narrow in its lower part, a narrow subcostal angle, and a bulging lower abdomen—the figure of the infantile type of visceroptosis. The abdomen was tender to pressure, especially in the epigastrium.

An X-ray examination which had recently been made by Dr. Swanson Hawks showed an hour-glass stomach (*Fig. 266*) and marked ptosis of the stomach and large intestine. The hepatic flexure was low, and the first part of the transverse colon passing down to the pelvis could not be manipulated free of the cæcum (*Fig. 267*). The middle part of the transverse colon lay in the pelvis looped on itself (*Figs. 267, 268*). There was considerable large bowel delay.

**OPERATION.**—The small intestines were seen emerging from a large transverse opening in the gastrocolic omentum. Above this opening lay the hour-glass stomach, and in the pelvis was the transverse colon lying behind the small intestines. The great omentum had almost disappeared, being represented by a few tags of fatty tissue forming weak adhesions at two or three points in Douglas's pouch. These were easily broken down, and when the transverse colon was lifted up the small intestines passed back through the gastrocolic omentum and the transverse mesocolon without difficulty.

The lower sac of the hour-glass stomach had an active ulcer on its posterior wall eroding the pancreas. This part of the stomach, together with the constriction, was resected, and the operation completed by the anterior

Polya method. The short loop of jejunum thus supported the transverse colon. The remains of the great omentum were sutured to the gastrohepatic omentum and to the stomach in order to prevent the transverse colon sliding to right or left across the loop of jejunum.

When last heard of three months ago she was quite well. The constipation was relieved as well as the stomach symptoms.

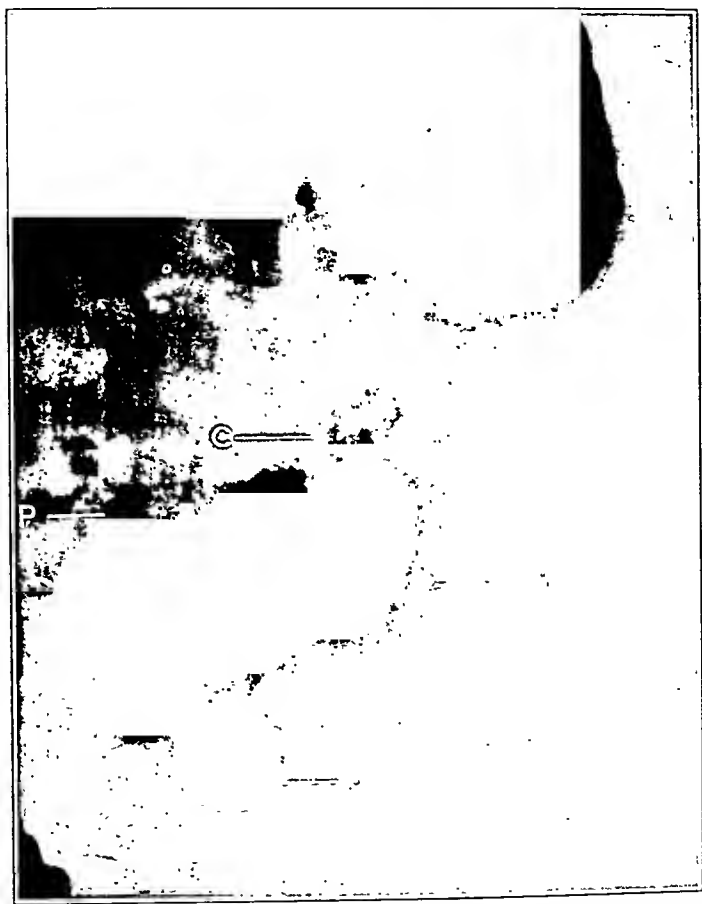


FIG. 266.—Case 2. Skiagram after opaque meal, patient horizontal, showing hour-glass stomach. A, Upper segment; B, Lower segment; C, Constriction; P, Pylorus; U, Umbilicus.

Of the above 21 cases of hernia of the small intestines through the transverse mesocolon, in 1 case the intestines emerged through the foramen of Winslow; in 8 cases the hernia passed out through the gastrohepatic omentum; and in 3 it passed the gastrocolic omentum; in the remaining cases the hernia stayed in the lesser sac. In 14 cases gastric or duodenal ulcer or carcinoma was present; in 2 there was organic hour-glass stomach, and in 2 others spasmodic hour-glass stomach. In at least 10 cases there was visceroptosis. Of 14 where we have been able to discover the sex, 13



were females and 1 only was a male. The ages, if we except Pringle's seventh case in a girl of 5, varied from 26 to 60 years.

The first of the two cases now recorded resembles Coffey's case in that an acute abdominal crisis seemed in each to mark the point of time at which the mesocolon gave way. The second is the only recorded clear case of the intestines emerging from the lesser sac through the gastrocolic omentum.

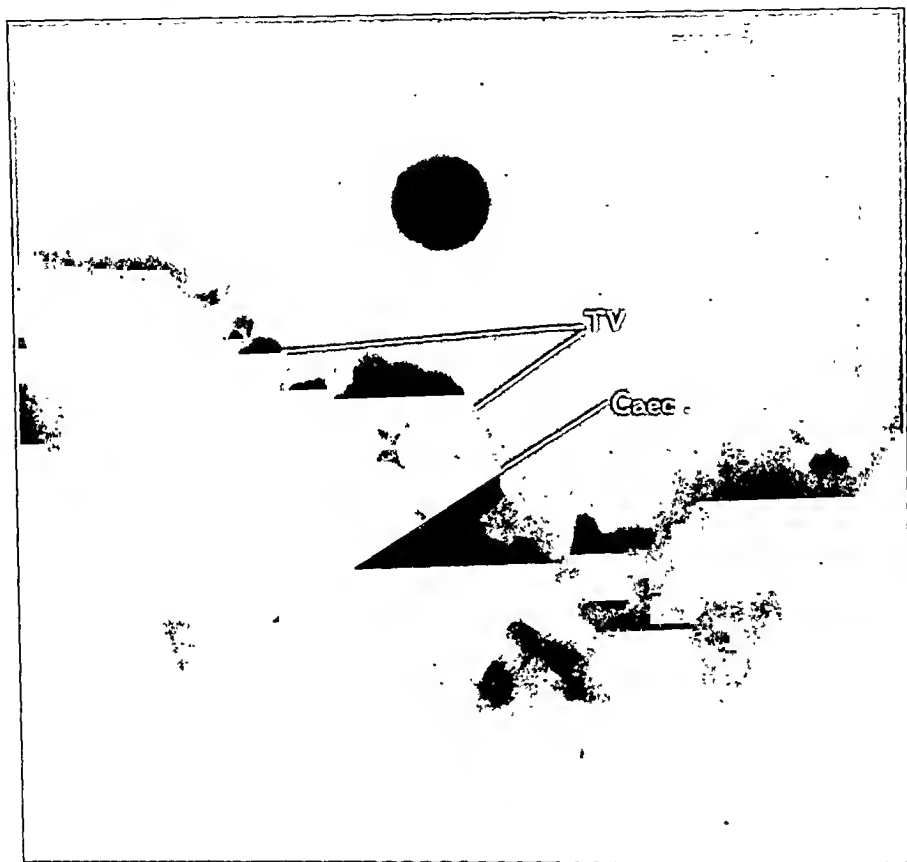


FIG. 267.—Case 2. Twenty-four hours after opaque meal, showing relation of caecum (Caec.) to transverse colon (TV) and looping of latter on itself. It was impossible by pressure to straighten out the loop in the colon or to raise it sufficiently from the caecum to be sure there were no adhesions. The stomach (S) still contained a trace of opaque meal.

Two other cases are mentioned in the literature, but the relations were uncertain owing to adhesions.

Apart from the rarity of our two cases the question of causation is of interest. Many causes have been suggested: retroperitoneal pouches, congenital defects of the mesocolon, violence, and ptosis. Pringle<sup>2</sup> and Chalmers<sup>6</sup> suggest that these herniae develop by way of the intermesocolic pouch, with secondary rupture into the lesser sac.

A case of intermesocolic pouch recently came under the observation of one of the writers. The anterior margin of the orifice was firm and unyielding, and capable of strangulating any gut within the pouch. We feel that if the 22 cases of hernia into the lesser sac had developed by secondary rupture from this pouch a number of cases of strangulation would also be on record.

The strength or weakness of the transverse mesocolon cannot be of much importance. Although fenestration is rare, the structure is at best flimsy,



FIG. 268.—Case 2. Seventy-two hours after opaque meal. The lower bowel had been cleared by enema. The transverse colon (TV) is still filled and looped on itself.

and incapable in itself of retaining the small intestines in their proper compartment of the abdomen. Security depends on equality of pressure on either side of this membrane.

It is generally recognized that mid-line ptosis predisposes to ulcer and hour-glass stomach. We consider it the all-important factor in the production of hernia through the transverse mesocolon. Coffey noted it in one case, Mayo in two; in fact it was definitely present in 10 of the 21 cases, and the remaining cases were females of an age and with a history at least consistent with the presence of visceroptosis.

It is easy to visualize the mechanism of the hernia. In the normal abdomen the relationship of the stomach and colon on the one hand and the small intestines on the other is such that when there is a sharp rise in intra-abdominal pressure, as, e.g., in vomiting, the stomach and colon are thrust more firmly into the upper compartment of the abdomen, and there support the transverse mesocolon against the upward pressure of the small intestines. If the stomach and colon are prolapsed, not only is this support lacking, but the weight of these viscera crowds the small intestines upwards under the stretched transverse mesocolon and increases their upward thrust in expulsive efforts—with possible rupture.

The direction which the small intestines will take on reaching the lesser sac will be determined by such considerations as the length of the gastrocolic omentum, the presence or absence of adhesions on the posterior aspect of the stomach, and the extent to which the stomach itself has prolapsed.

Two further points deserve mention: (1) Seeing that mid-line ptosis so commonly accompanies hour-glass stomach, in dealing with the latter there is a *prima facie* reason for preferring the anterior to the posterior operation (whether Polya or gastrojejunostomy), as in the anterior method the prolapsed colon is supported by the jejunal loop; (2) It is probable that hernia into the lesser sac arising after gastro-enterostomy occurs only in the presence of ptosis.

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- <sup>1</sup> SHORT, A. RENDLE, *Brit. Jour. Surg.*, 1923, Jan., xii, 456.
- <sup>2</sup> PRINGLE, J. HOGARTH, *Glasgow Med. Jour.*, 1919, xci, 129 (Case vii).
- <sup>3</sup> COFFEY, *Surgical Monographs: Gastro-enteroptosis*, 1923, 208.
- <sup>4</sup> CRYMBLE, P. T., *Quain's Anatomy*, 11th ed., ii, part 2, Fig. 349.
- <sup>5</sup> STOLZENBERG, *Virchow's Arch.*, 1910, ccx, 470.
- <sup>6</sup> CHALMERS, *Jour. Pathol.*

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## AN UNUSUALLY LARGE TUMOUR OF THE RIGHT BREAST.

By W. E. TANNER, LONDON.

THE patient, a married woman, age 53, who had had ten children, was seen in September, 1924. She said that the right breast had been gradually getting larger for seventeen months, that during the last month it had rapidly increased in size, and during the last week there had been much watery discharge from the right nipple.

ON EXAMINATION.—A stout woman of healthy appearance. In the right breast was a large tumour apparently arising in the upper part of the breast (Fig. 269). There were irregular rounded projections springing from the tumour, particularly in its upper part. The nipple was not retracted, and, with the lower part of the breast, was pushed downwards and forwards by the tumour. The skin was not attached, and was normal in appearance except over the upper part of the tumour, where it was thinned by pressure and the fluctuating projecting masses appeared blue through the thinned skin. The tumour was freely movable on the chest wall, and no enlarged glands could be felt in the axilla or above the clavicle. The mediastinum

was clear, and nothing abnormal was detected in the other systems of the body.

A diagnosis of Brodie's serocystic tumour of the breast was made; the patient was advised to have the right breast removed, and a good prognosis was given. The breast was excised on Sept. 25, and weighed 18 lb. She made a good recovery and has had no complications of the breast condition. In March, 1925, a large gall-stone was removed from the gall-bladder.

DESCRIPTION OF THE TUMOUR.—I am indebted to Mr. T. H. C. Benians for the following description of the macroscopical and microscopical structure :—

"The tumour is rounded, with irregular rounded bulging areas mostly at the upper part. The skin is normal in appearance for the most part, but thinned and bluish over some of the upper projecting masses; it moves freely over the whole tumour. The nipple is slightly projected, but is not adherent.



FIG. 269.

"A vertical section through the middle of the tumour shows a thin layer of fat and subcutaneous tissue between skin and tumour; the tumour bulges freely towards the cut surface, especially in its central parts. Towards the skin surface, and in most parts immediately at the margin of the tumour, is a deep cleft bounded by a thickened membrane which is apparently a cyst wall with numerous cyst cavities opening out of it, so that the bulk of the tumour is lying inside a multilocular cyst. The cyst wall is white and tough, and in parts covered by thickened white and yellowish patches. The cyst also contains whitish-yellow semi-fluid and granular material, which is made up of cholesterin and degenerate matter. The tumour projecting into the cyst is smooth and white or pinkish on section, but on its free surface, which is nodular, it has a clear mucoid appearance in parts, in other parts an opaque white almost cartilaginous look, although soft to cut, and other parts are

covered with dilated capillaries. No large vessels are to be seen, and there are no hæmorrhages.

"Sections cut from various parts of the tumour present a varying histological picture. The bulk of the tumour which formed the intra-cystic masses is composed largely of spindle cells, and these vary greatly in type and in arrangement. In parts they are large, deeply staining, and fitted closely against each other; the nuclei of these cells are relatively large in comparison with the cytoplasm. In parts the cells are more elongated, with rod-shaped nuclei, and are more of the type of adult connective-tissue cells. In a still further variation the cells are myxomatous in type, with small or larger clear areas between them; this myxomatous change affects a large part of the tumour. Irregularly interspersed throughout the mesoblastic tissues are gland tubules lined with a cubical epithelium, and in places these are dilated into small cysts. In some parts, and more particularly in areas showing gland tubules, are numerous irregular structureless areas apparently derived from a hyaline degeneration of the tumour cells. Blood-vessels are scarce but well formed.

"Sections of the small part of breast tissue outside the tumour show an increase of fibrous tissue and some lobular hyperplasia. The fibrous tissue is more dense towards the free surface where it constitutes the cyst wall. The epithelial lining covering it varies in different parts from a single layer of cubical cells to a stratified layer half a dozen cells in thickness; these epithelial cells are largely degenerate and show no nuclei."

Dr. G. W. Nicholson, Morbid Histologist to Gny's Hospital, who very kindly examined the sections, reports as follows: "The sections examined were from the edge of the tumour and show the structure of a fibro-adenoma, the connective-tissue matrix of which is composed of whorls of spindle cells, some of which have undergone hyaline change. They surround spaces lined by glandular epithelium. The surrounding breast is compressed, and forms a capsule for the tumour."

In the *Medical Times* for 1844 Sir Benjamin Brodie gives a description of two similar cases. He says that the disease is often confounded with carcinoma, and is not met with in hospital practice, but often shows itself in private life. In the first patient the tumour was about the size of a walnut. It was punctured, found to contain serum, and then laid open with a lancet. A year later a fungous tumour was found where the cyst had been opened. The breast was amputated, and was made up of cysts containing fluid matter. A solid tumour projected from the inner surface of the largest cyst. In the second case the breast on removal was found to weigh between 7 and 8 lb. He states that the skin does not always ulcerate in these cases, but occasionally it is so distended that it bursts. In conclusion he says: "I have given no name to this affection, because I think it is an error of modern times to be continually giving new names to diseases, but if it must have a name I think it should be called serocystic tumour."

## REVIEWS AND NOTICES OF BOOKS.

**Clinical Researches in Acute Abdominal Disease.** By ZACHARY COPE, B.A., M.D., M.S. (Lond.), F.R.C.S. (Eng.), Senior Surgeon to Out-patients, St. Mary's Hospital; Surgeon, Bolingbroke Hospital, etc. Demy 8vo. Pp. 148, with 31 illustrations. 1925. London: Oxford Medical Publications. 12s. 6d.

THE work consists for the most part of an expansion of the Arris and Gale Lecture, 1922, publications in the *Lancet*, 1924, and in the BRITISH JOURNAL OF SURGERY and *British Medical Journal*.

Chapter 1 deals with the scope of clinical research, and the author suggests that in clinical research it is not necessary for two workers to collaborate as it is in experimental work from a laboratory, as the means of verification of opinions offered in clinical research are near at hand for everyone to test. Since reading this book, and on many occasions before, we have applied the suggestions put forward and have found them of the greatest possible help in many cases. The three main purposes to which clinical research may be directed are: (1) It is within the scope of the clinician to test with thoroughness the results achieved by work in the laboratory. (2) It is within the scope of clinical research to try various empirical methods, and to furnish the results to the laboratory for explanation and elaboration. (3) It is within the scope of clinical research also to record and compare clinical facts with the object of determining some questions which are inaccessible to laboratory methods of study. The author gives five rules which he says should be observed if clinical research is to be of any value.

In the chapter on the function of the parietal peritoneum two areas of peritoneum are described; one, the demonstrative area, which includes all the lining of the abdominal cavity except the pelvis and the central and inferior part of the posterior abdominal wall; and the other, the non-demonstrative or silent region, which comprises the pelvis and that part of the posterior abdominal wall bounded by the ascending colon, descending colon, and the transverse mesocolon. It is suggested that the non-demonstrative area is probably supplied via the sympathetic system and the demonstrative from the main somatic nerves. Stimulation of the non-demonstrative areas does not give rise to any clinical localizing symptom or sign. For this reason an acutely inflamed appendix either in the pelvis or behind the lower end of the ileum may give rise to no sign of irritation, and may be present with entire absence of muscular rigidity. A point which should be thoroughly realized is that rigidity of the abdominal wall is not necessarily present in acute appendicitis, and that when this is not present the appendix is usually found in the pelvis or behind the lower end of the ileum.

Chapter 4 is illustrated by drawings of areas of hyperæsthesia found in acute appendicitis, perforated gastric and duodenal ulcers, cholecystitis, etc. It cannot be taken as an indication of the existence of any particular lesion, but of an irritative condition being present in the area supplied by that particular segment of the cord.

Phrenic shoulder-pain is described at length, its presence in acute abdominal disease, and its usefulness as a method of differential diagnosis between disease in the abdomen and in the thorax. Chapter 6 is devoted to the differential diagnosis of abdominal and thoracic disease, and a general summary of the different signs and tests is given in a comparative table.

A particularly interesting chapter is that dealing with the differential diagnosis of genito-urinary symptoms in acute appendicitis. There is no doubt that innocent appendices have been removed in many cases of stone in the right ureter.

A new test—the femoral test—is described in Chapter 7: inflammation of the peritoneum over the external iliac vessels causes pain in the hypogastric region

when pulsation in the femoral artery is stopped by pressure in the thigh; this sometimes proves of value in determining the position of an acutely inflamed appendix.

The book is well arranged, and the drawings are simple and clear. Every chapter contains suggestions of great interest which will undoubtedly help the surgeon in differential diagnosis. This latter is a subject which we think needs emphasis, as many are inclined to be satisfied when they have decided whether an abdomen should be opened or not. For those who take a deeper interest in their work this book will prove of the greatest help.

**Minor Surgery.** By LIONEL R. FIFIELD, F.R.C.S., Surgical First Assistant and Registrar, London Hospital; Demonstrator of Minor Surgery, London Hospital. Crown 8vo. Pp. 431 + x, with 273 illustrations. 1925. London: H. K. Lewis & Co. Ltd. 12s. 6d. net.

IN this book the author has endeavoured to give an account of minor surgery as taught at the London Hospital, and he is to be congratulated on the result, which does great credit not only to himself but also to the London Hospital Medical College.

Surgery, wherever it is taught, should be essentially the same, though details may vary according to individual taste or custom; and whether major or minor surgery be considered, the great underlying principles cannot alter or vary. Teaching, especially the teaching of students in their earlier years, should be systematic, though it is well for them as the time comes for qualifying and assuming responsibility that they should have a knowledge of the practical experience of their seniors, so that they may have some guide to the most efficacious forms of treatment and be able to give a reasonably accurate prognosis in the cases they are likely to meet in practice. This little book, under the modest title of *Minor Surgery*, treats of the great principles as well as the details of minor operations; and, while being systematic to a degree, gives valuable advice in the choice of alternative methods of treatment and also in the results which may be expected from them.

A great deal of material of doubtful value which used to appear in book after book has been omitted, and this has enabled the author to emphasize the things he considers most important, while confining himself strictly to surgical conditions which, however great their importance, could not be termed major surgery. To discriminate between major and minor must often have been difficult, but the author's judgement has been well considered. He refers frequently to his own experience, but also quotes freely from the writings and practice of well-known surgeons whose work has enriched the science and art of surgery in recent years, thus bringing the book absolutely up to date.

The early chapters give an account of the routine examination of a patient, the acute abdomen, acute intestinal obstruction, antiseptic and aseptic technique, and the methods of sterilization of instruments and materials. Preparation for operation and operative technique are well described; and after-treatment, including the treatment of shock and other complications, is then considered. A slight error here is that massage is mentioned in the treatment of venous thrombosis with insufficient warning.

We then pass to chapters on wounds, hæmorrhage, cellulitis, and abscess, all of which are full of sound practical teaching. Chapter 7, on infection of the hand, gives a résumé of the work of Kanavel, to whom the author pays tribute. The section on bandaging is useful in that it includes only the methods most commonly used. The chapter on minor operations contains an astonishing amount of information and is well illustrated. The section on fractures is admirable, because it includes an account of the after-treatment of the fractures of each bone, and the prognosis and probable period of incapacity. With the dislocations is included a brief but satisfactory account of internal derangements of the knee-joint.

The chapters on diseases of joints, genito-urinary affections, and trusses, and the short chapter on the eye, require no comment except that they maintain the standard of the earlier parts of the book, which ends with a chapter on the administration and choice of anæsthetics. Apart from a number of printer's errors the book is well produced, and it can be confidently recommended as being of value in the teaching, study, and practice of minor surgery.

**Some Encouragements in Cancer Surgery.** By G. GREY TURNER, F.R.C.S., Hon. Surgeon, Royal Infirmary, Newcastle-upon-Tyne. Pp. 75, with 40 illustrations. 1925. Bristol: John Wright & Sons Ltd. 7s. 6d. net.

THIS little book is a record of some cases of malignant disease operated on by the author, and contains forty excellent illustrations of the specimens removed. The cases are examples of cancer from all the common sites, and many are instances of advanced disease. Mr. Grey Turner has followed up these cases in order to determine the length of life after operation and the cause of death, and the book has been written to encourage surgeons to persevere in efforts to relieve, and at times to cure, patients suffering from what appear to be, in many instances, very unfavourable growths from the operative point of view.

He is fully alive to the advantages of operating in the earliest possible stage of the disease, but he points out, and we think rightly, that a long history often means a good resistance on the part of the patient, and that a formidable lump, under these circumstances, does not necessarily deprive the patient of the possibility of being cured by a sufficiently determined effort on the surgeon's part. There is an inclination in the minds of quite a number of the profession to regard all operations for malignant disease as doomed to failure, irrespective of the stage at which the patient is seen, of the fight he may have put up against the disease as evidenced by the duration of the symptoms and the general well-being in spite of a large local lesion, and of his desire to take what is, after all, his only chance of getting rid of the disorder. We recognize that this gloomy outlook is not seldom justified by events, but we recommend those who suffer from this hopeless attitude of mind to read this book of Mr. Grey Turner's, and to revise their antipathy to operation, for they will find reliable records of patients living many years who would inevitably have been dead of their disease years before, and moreover who, when they did die, succumbed to something quite unconnected with the cancerous growths for which the operations were performed.

Mr. Grey Turner says in his book that in his opinion every case which does not show unequivocal evidence of dissemination should have the chance which only an operation offers, and in our view this is the only justifiable advice to give patients suffering from this disease at the present time. It is the unanimous opinion of all those who know anything about malignant disease that it is in the first place a local lesion, and probably remains so longer than we think, and that it can always be removed entirely if the attempt be made before dissemination begins. It is only by adopting an optimistic attitude towards the operative treatment of malignant disease that we, as a profession, can hope to induce patients to come earlier and yet earlier for treatment, and this is the key to the situation from the curative point of view.

If Mr. Grey Turner's book does anything towards converting the pessimists of the profession it will have achieved a most useful purpose, and we feel that a careful study of the cases recorded cannot have any other effect on an unbiased mind.

**Cancer and the Public: The Educational Aspect of the Cancer Problem.** By CHARLES P. CHILDE, B.A., F.R.C.S., M.R.C.P.E.; President B.M.A., 1923; Chairman of Public Health Committee, Portsmouth; Consulting Surgeon, Royal Portsmouth Hospital. Demy 8vo. Pp. 267. 1925. London: Methuen & Co. Ltd. 10s. 6d. net.

THIS book is intended both for the general public and for the medical profession. In an introductory chapter the author adduces good reasons for thinking that the subject of cancer and what can be done for it by timely and suitable surgery should be brought prominently before the public. Whatever view may be taken of this debatable subject, it is well that a book of this kind should be written by one who has wide knowledge and ripe experience and who is capable of clear reasoning and cool judgement in this important matter.

The book deals solely with carcinoma, and especially with carcinoma occurring in certain definite situations, and with the prospects of its cure by surgical removal. Sarcoma is excluded from the scope of the book.

In an amusing and well-written chapter on cancer theories, the author discusses briefly the numerous theories that have been brought forward, both by the profession and by the laity. He shows how shallow are the arguments by which most of



them are supposed to be supported. He pours quite justifiable ridicule on the idea that articles of diet, or intestinal stasis, or lack of vitamins, are likely to have anything to do with its production. He does not consider that climate, locality, occupation, or mental worry, all of which have their advocates, have anything to do with the causation of cancer. Although rightly laying stress on local irritation as a frequent determining cause, he wisely concludes by saying that the "riddle of its cause has not been solved".

Various cancer fallacies held by the public are then discussed, followed by seven prominent 'facts', at five of which no surgeon is likely to cavil. The first and third, however, although possibly true, can scarcely be said to have been proved. The inferences to be drawn from these facts are then related. The curability of cancer, the condition of cure, and the possibilities of cure are then ably and temperately discussed, with abundant illustrative statistics.

What the public should know about cancer, its danger signals, and the means to be taken for its cure, are dealt with at some length. The American campaign against cancer is contrasted with the apathy shown on this side of the Atlantic. A more thorough education of the public on the known facts of cancer and the hopefulness of cure in cases which are seen early is strongly advocated.

After a short chapter on radiotherapy, its limitations and uses, the author concludes very sensibly by saying that his object has been to "draw attention to the educational aspect of the cancer problem; to brush away former errors; to show that cancer is capable of being dealt with successfully by surgical operation at the present time, and that it is the only method we possess of treating it hopefully; but that the very condition of success lies in that knowledge on the part of its victims which will enable them to avoid delay in seeking advice. My purpose has been to demonstrate that until surgeons are enabled to come face to face with cancer in its earliest beginnings, its treatment by operation must remain generally ineffectual, only exceptionally curative; that education is essential to and must precede successful treatment; that as long as people apply as they do now for the first time with advanced cancer, so long will the results of its treatment spell failure" (p. 258).

A blemish in what is otherwise a good book is the unnecessary, and, as many will think, the totally unwarranted comparison of cancer with acute appendicitis (pp. 111, 113, 189). To say that a patient with an acute perforated appendix unless immediately operated upon cannot avoid "certain death" is at least a gross exaggeration. Many thoughtful and experienced surgeons and physicians at the present day know better than this. In any case, even if the statement were true, it has no bearing on cancer, and had better have been omitted, especially as it is likely to cause the well-informed reader to suspect, although quite undeservedly, the otherwise cool judgement of the author.

The book is well written and is easy to read, although there is perhaps a little too much repetition in places. For example, on p. 154, we read again much of what has already been said on p. 4. Misprints are very few, but there is an obvious one in a Latin quotation on p. 113, and another on p. 149 in the German title of a book, which, however, is correctly spelt in the table of references on p. 261.

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**Fractures and Dislocations: Immediate Management, After-care, and Convalescent Treatment, with Special Reference to the Conservation and Restoration of Function.** By PHILIP D. WILSON, M.D., F.R.C.S., Instructor in Orthopedics, Harvard Medical School; and WILLIAM A. COCHRANE, F.R.C.S. (Edin.), Tutor in Clinical Surgery, Edinburgh University. Pp. 789 + xv, with 978 illustrations. 1925. London: J. B. Lippincott & Co. 45s.

This is certainly one of the best works on fractures and dislocations which has appeared in recent years. Not only is it the joint work of an American and a Scotchman, but it also represents the generally accepted principles of treatment of the members of the staff of a large general hospital (Massachusetts). In this institution all fractures are looked after by a special 'Fracture Service', consisting of six surgeons, four of these being 'general' surgeons and two being orthopædic. This ideal arrangement, the adoption of which is long overdue in the hospitals of

this country, makes it possible to come to some general agreement about the most practical and successful methods of treatment. The present work represents the outcome of such an agreement.

Ample space is devoted to general considerations, and especially to those of anatomy and physiology. The treatment of each type of injury is described in a very practical manner, and followed up until complete functional restoration has been achieved. Methods of splinting are reduced to the simplest possible terms, full value being ascribed to the various patterns of Thomas and Jones, and also to plaster-of-Paris and traction devices. The book is intended mainly as a guide to the student and general practitioner, and therefore most stress is laid upon non-operative methods. The indications for open operation are mentioned—perhaps with a rather conservative bias—but the details of operation are omitted.

In addition to those on fractures and dislocations of the limb bones and joints, there are chapters dealing with fractures of the spine and of the facial bones. The former is very valuable, the latter less so.

The great value of the book consists in its wealth of practical details, all of which represent the considered opinion of a large group of experienced surgeons. The profuse illustrations greatly help the elucidation of the text; in fact the figures alone are almost a complete treatise. The chief criticism of the work is that the open operations are put too much in the background, and even when mentioned are not described.

**The Principle of Early Active Movement in Treating Fractures of the Upper Extremity.** By J. W. DOWDEN, M.B., F.R.C.S.E., Surgeon to Chalmers Hospital; late Surgeon to the Edinburgh Royal Infirmary. Large 4to. Pp. 111 + xvi, with numerous plates. 1924. Edinburgh: Oliver & Boyd. 16s. net.

This volume consists of a number of reproductions of X-rays and photographs of fractures of the arm in various stages of treatment. The letterpress is sketchy, and serves to do little more than explain the various pictures. The method certainly contravenes a well-accepted principle, that immobilization should be continued until callus is sufficiently strong to maintain the fragments in order to restore positions. It appears from the letterpress that the author pays no particular attention to the reduction of displacement by manipulation under an anaesthetic and checked by X rays; yet, as far as can be judged in the notes, the treatment seems to be followed by good functional results; how large a part in this is played by the persuasive personality of the author or by the hardihood and insensibility to pain of his Edinburgh patients, this is not the place to judge. It is possible—in fact, to the reviewer probable—that Mr. Dowden claims too much for this line of treatment; but we would agree with him that there is much to be said for the use of active movement in the after-treatment of fractures, just as there is much to be said against the use of passive movements, and if this volume directs attention to this principle it can only do good.

The reproductions are well done, but the author falls into a common mistake in describing various radiograms as “separations of the lower humeral epiphysis” which are really those of supracondylar fractures.

**Anæsthesia.** By JAMES TAYLOR GWATHMEY, M.D., First President of the American Association of Anæsthetists; with collaborators on special subjects. Second revised edition. Royal 8vo. Pp. 799 + xxv, with 273 illustrations. 1924. London: J. & A. Churchill. 25s. net.

In his new edition Dr. Gwathmey has produced a comprehensive account of the speciality as practised to-day; owing to recent progress, he has found it necessary to delete chapters on such subjects as electrical anæsthesia and statistics in order to make room for more important and practical material. In the introductory section the history of anæsthesia is fully and delightfully told. In the second chapter the physiology of the various methods is put shortly but exceedingly clearly. The author has entrusted the description of some of the subjects to a number of collaborators who are recognized authorities on the methods concerned, such as Elsberg on intratracheal insufflation, and so forth.

Dr. Gwathmey's capacity for combining the practical with the theoretical has enabled him to produce an exceptionally reliable chapter on each of the standard anæsthetics—chloroform, ether, gas, and ethyl chloride, and the reader is left with a clear mental impression as to their respective uses and modes of employment.

Local anæsthesia has been allotted a quite prominent position in this book, and in referring to the advantages to be derived from combining local with general anæsthesia the author seems to suggest that there is room for improvement in the training of the surgeon for his share in this valuable practice.

The book is well got up and efficiently illustrated, and cannot fail to become a favourite with specialist and student.

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**Local Anæsthesia: its Scientific Basis and Practical Use.** By Prof. Dr. HEINRICH BRAUN, Zwickau. Translated and edited by MALCOLM L. HARRIS, M.D., Professor of Surgery, Chicago Polyclinic. Second American from the sixth revised German edition. Royal 8vo. Pp. 411 + xxi, with 231 illustrations in black and colours. 1924. London: Henry Kimpton. 25s. net.

IN view of the widespread and increasing interest in and employment of local anæsthetics, the appearance of another edition of this comprehensive standard text-book is most welcome.

The first eight chapters deal with the theoretical side of local anæsthesia, and include a detailed account of the characters and properties of the various drugs which may be used for its production. A notable addition to this section is a fuller treatment of the subject of poisoning by novocain. Chapters 9 and 10 give a general account of the technique of the various methods of application of local anæsthetics, and a useful section on their value and indications for their use. There is a very well illustrated description of lumbar and sacral anæsthesia, which is one of the features in which this edition excels its predecessors.

The remaining half of the book is devoted to descriptions of the exact method of inducing anæsthesia for operations on the head, neck, trunk, and extremities. In fact, extremely few of the operations of surgery are omitted, so that the possibilities of local anæsthesia are very fully set forth. We cannot but remark that although almost any major surgical procedure may be carried out under this form of anæsthesia, yet its application must be limited by the individuality of the surgeon and the patient. The illustrations, in black and colours, are excellently clear, but even they emphasize the fact referred to above—that the 'strain' upon patient and surgeon must be exceedingly severe in some cases, and that what is possible under local anæsthesia may not always be quite advisable.

For those who wish to explore the field of local anæsthesia, this book may be recommended as giving a comprehensive description of the subject, with large numbers of references to the practical experience of the author and other writers.

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**The Technic of Local Anæsthesia.** By A. E. HERTZLER, A.M., M.D., Ph.D., LL.D., F.A.C.S., Professor of Surgery in the University of Kansas. Third edition. Imp. 8vo. Pp. 272, with 140 illustrations. 1925. London: Henry Kimpton. 25s. net.

A COMPREHENSIVE work describing in detail the technique for the employment of local anæsthesia in almost every operation that the surgeon is called upon to perform. A general review of the whole subject is followed by a detailed account of the more commonly undertaken operations. The directions given are simple and easy to understand, the plan followed being to describe briefly what the surgeon sets out to do and how he can best carry it out under local anæsthesia. Some of the more ambitious procedures are dismissed briefly, and it is pointed out that the simpler operations should be mastered, and then as the surgeon gains confidence in himself the more difficult ones can be added to his list. If he is to be successful, the operator must have perfected his routine by experiment on the cadaver before he employs it on a patient. Stress is laid on the giving of an occasional word of encouragement to a patient who is undergoing a great mental strain.

Local infiltration and field blocking are described as the methods of choice, being preferred to paravertebral anæsthesia or the blocking of individual nerves,

the reasons given being that they are more certain in their action and that they may have to be employed to supplement the latter procedures when they are used.

The use of quinine is advised on a more extensive scale than has hitherto been practised, and from the author's experience seems to merit a more prolonged trial than has been accorded to it in the past.

Throughout the book the style is good, the description clear and simple, and altogether it is to be recommended to those who desire a small handbook dealing with the whole subject of local anaesthesia.

**The Westminster Hospital Reports.** Vol. XIX, 1924. Edited by E. ROCK CARLING and HILDRED CARLILL. Pp. 299 + xxvi, illustrated. London: Henry J. Glaiser. 7s. 6d. net.

THE *Reports* of the Westminster Hospital have been discontinued for ten years, and this is the first of the new volumes. It was hoped to present a systematic study of the post-mortem records for the last ten years; but such a plan was found to be impossible, and the present volume consists of a haphazard collection of individual contributions, only the first six of which form part of the original concerted plan. Sir Edward Pearson contributes a foreword, in which he expresses an opinion favourable to the efficiency of the Voluntary Hospital system. On the face of it, it would appear that the haphazard appearance of scientific reports would hardly be the occasion for this tribute.

The book, though small, is full of interest from cover to cover. As a kind of celebration of the rebuilding of the hospital, Mr. Spencer has written a most fascinating section about the history and growth of the old institution. Then follow six chapters devoted to the post-mortem records relating to special subjects. Mr. Spencer writes the section dealing with the mouth and tongue, pointing out the importance of pulmonary complications in causing a fatal issue. Mr. Turner deals with gastric and duodenal ulcers, of which there had been 370 cases, with a mortality of over 10 per cent, the duodenal cases having a higher mortality than the gastric. Mr. Pinto Leite writes on the post-mortem evidences of lung complications due to general anaesthesia, and he finds no evidence that ether or closed ether is more dangerous in respect to its effect on the respiratory apparatus than other anaesthetics. Mr. Braxton Hicks analyses the cases which came before the coroner; Dr. Goodwin discusses the toxæmias of pregnancy; and Mr. Rock Carling describes 19 cases of fatal pulmonary embolism. The rest of the volume consists in short papers and in statistical tables.

**Guy's Hospital Reports.** Vol. LXXIV (Vol. IV, Fourth Series), No. 4, October, 1924. Edited by ARTHUR F. HURST, M.D. Medium 8vo. Pp. 121, with 49 illustrations. 1920. London: Wakley & Son. 12s. 6d. net.

*Guy's Hospital Reports* continues to provide a number of papers from members of the staff, and the present number contains observations on cases of Addison's disease, articles on methæmoglobinæmia, arachno-dactyly, and several other subjects. There is a paper on deep X-ray therapy, from which one hoped it might have been possible to have obtained some accurate knowledge of the value of this treatment; presumably this is too early to be expected, for the article deals almost entirely with the details of technique, and not with results.

**A Synopsis of Surgery.** By ERNEST W. HEY GROVES, M.S., M.D., B.Sc. (Lond.), F.R.C.S., Surgeon to the Bristol General Hospital; Professor of Surgery, University of Bristol. Crown 8vo. Pp. 671 + viii, with 164 diagrammatic illustrations. 1925. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

THIS seventh edition, enlarged and revised, now contains 671 pages and many line illustrations. It is a synopsis of surgery, as the Preface states, but this synopsis is now becoming as large as the text-books themselves of thirty years ago. The fact that this work has gone through seven editions is sufficient guarantee that it is wanted, and though some teachers do not see eye to eye with the author in this method of imparting instruction, it is clear that it is a book that the student wants and will have.

**Normal Bones and Joints Roentgenologically Considered.** By ISIDORE CONN, M.D., F.A.C.S. Pp. 218 + xxxii, with 251 illuſts. 1924. New York: Paul B. Hoeber. \$10.

THIS is the fourth volume of a ſeries published as the *Annals of Roentgenology* under the editorſhip of James T. Caſe, M.D. It opens with a foreword by Rudolph Matas, which deſcribes in a moſt intereſting manner the growth of our knowledge of bones in their ſtructure and development which has ſprung from Roentgen's diſcovery of 1895. The work itſelf is admirable in its ſimplicity and clearneſs. For the moſt part it deals only with the normal bones at their articular extremities. There are ſix chapters devoted to the main joints of both upper and lower extremities. Each chapter begins with a ſeries of extracts from ſtandard works on anatomy and ſurgery, giving the current views about the development of the epiphyſes. Then follow excellently reproduced X-rays of the joint in queſtion, taken at various ages from infancy up to adult life. Theſe are of the utmoſt value in giving definite evidence about the centres of oſſification and their dates of fuſion, and alſo in providing reference plates with which any given clinical condition may be compared. Only in connection with the elbow-joint is any detailed reference made to traumatic conditions. We would ſuggeſt that it would be of the utmoſt value if typical injuries were figured of all the regions deſcribed, and alſo if the varying normal conditions of the ſhafts of the bones, as well as their extremities, were given at different periods of life, eſpecially thoſe of old age. The book will be an invaluable work of reference.

**Diagnostic et Traitement des Maladies de la Vésicule biliaire par l'Excrétion vésiculaire provoquée.** By M. CHURAY and M. MILOCHÉVITCH, Paris. Crown 8vo. Pp. 156, with 13 illuſtrations. 1924. Paris: Maſſon et Cie. Fr. 12.

A NUMBER of teſts have been deviſed whereby a flow of bile is excited and the reſulting fluid collected through a duodenal tube. In this book the theoretical baſis and the practical application of ſuch teſts are diſcuſſed. Stimulation of the duodenal mucoſa by 30 per cent magnesium ſulphate (Meltzer-Lyon teſt) is the method moſt under conſideration, but the uſe of 5 to 10 per cent peptone (Stepp's teſt) and ſulphuric ether (Katsch teſt) is alſo reported on.

The authors have dealt ſystematically with the ſubject in its different aſpects. A full account of the technique of paſſing a duodenal tube is given, together with the means by which the ſtages of its progreſs to its final ſite may be obſerved and verified. The difficulties that may ariſe, both in paſſing the tube and in obtaining pure ſamples of the fractions of bile, are indicated. A ſection of the book is devoted to the hypotheſes concerning the derivation of the fractions of bile, more eſpecially the ſecond, which is conſidered by ſome to come from the gall-bladder and by others to be an increased flow direct from the liver. Further ſections deal with the diagnostic value and the therapeutic value of the teſts. The book concludes with a réſumé of the more recent work on the ſubject.

**La Transfuſion du Sang.** By P. EMIL WEIL and PAUL ISCH-WALL. Pp. 248, with 18 illuſtrations. 1925. Paris: Maſſon et Cie. Fr. 20.

THIS work conſtitutes a complete monograph on the ſubject of blood tranſfuſion, and though there is little in it which has not already been written by other authors, the book is one which we can thoroughly recommend to the profeſſion. It is eſſentially a practical treatiſe, and though theoretical conſiderations are, here and there, introduced, they do not bulk ſo largely as to confuse the reader and diſtract his attention from the proved therapeutic value of blood tranſfuſion.

The authors inſiſt on the ſimplicity of the operation, and alſo the ſafety of its employment when the bloods have been found to be compatible, and urge its extended uſe, eſpecially at an earlier ſtage, in whatever condition it is indicated. With this view we entirely agree. as, with the authors, we feel ſure that the fancied difficulties of technique, the ſuppoſed dangers ariſing from mixing the bloods of different people, and the poor reſults ſo often attained with blood tranſfuſion from uſing it as a laſt reſource, are reſponsible for the tardy acceptance of this eſſentially physiological method of treating many ſurgical and medical complaints.

The authors state that all the indications are not as yet definitely fixed; however, accumulated experience permits them to say that imperative indications are hæmorrhages of all kinds, shock, and anæmic states. In many other conditions beneficial results will be attained, though good will not always follow, especially if only employed late in the disease, viz., puerperal infections, pre-operative and post-operative therapy, pernicious and other forms of anæmia, etc.

There are ten chapters, headed as follows: Historical; Biology of transfusion; Citrated blood; Accidents; Methods of preventing accidents; Technique; Surgical indications; Obstetrical indications; Medical indications; Transfusion of blood in the infections; Conclusions. The book is terminated by a comprehensive bibliography, which is arranged in such a way that there can be no difficulty in finding the desired reference, as it is also tabulated under the same headings as the chapters in the book.

*Les Hépatites dysentériques et leur Traitement.* By A. VALASSOROULO and PAVLOS PETRIDIS. With a Preface by Dr. E. RIST. Royal 8vo. Pp. 145 + viii, illustrated. 1924. Paris: Masson et Cie. Fr. 12.

THIS book is based on experience gained in the Greek Hospital at Alexandria, and should be of interest to all called upon to deal with this disease. It is divided into two portions. The first deals fully with pathology, symptomatology, and medical treatment. Treatment by emetine is discussed very fully. In the surgical portion, written by Pavlos Petridis, although other methods are discussed, pride of place is given to the operation introduced by his father, Aristide Petridis, which is fully described and illustrated. This is one of those books written as the result of personal experience that are always instructive.

*Traitement chirurgical de la Dilatation bronchique.* By P. GUIBAL, Paris. Deny 8vo. Pp. 173, with 31 illustrations. 1924. Paris: Masson et Cie. Fr. 10.

THIS small book actually may be taken as an abstract of the various methods of surgical treatment of bronchiectasis, and as such is most excellent. After a brief review of the clinical forms of bronchiectasis and the anatomical relations, the rest of the book is devoted to the subject of treatment. The major portion is connected with thoracoplasty and removal of one or more lobes of lung.

*Die Hyperostosen des Schädels.* By DR. INASABURO NAITO, with Introduction by PROFESSOR SCHÜLLER. Imperial 8vo. Pp. 95, with 84 X-ray photographs and 2 plates. 1924. Vienna: Julius Springer. 3.25 dollars.

THIS short monograph gives a clear and well-illustrated account of the various conditions in which local or diffuse thickening of the skull bones occurs. The conditions described include rickets, acromegaly, Paget's disease, leontiasis ossea, otitis fibrosa, exostoses, neoplasms, inflammatory thickenings including syphilis, together with traumatic and compensatory enlargements of the skull bones. Each group of cases is illustrated by a number of typical specimens, the structure and X-ray appearances of which are given in detail. This makes the book a very valuable work of reference. A short concluding chapter deals with the classification and differential diagnosis of these various conditions.

*The Medical Year Book and Classified Directory, 1925.* Edited by CHARLES R. HEWITT. Pp. 596. London: William Heinemann. 12s. 6d.

THIS is the second year of issue of this small guide to the medical profession in England, Scotland, and Wales. It gives a short account of the various medical authorities in this country, together with the Universities and teaching hospitals and their staffs. No doubt it will be useful as a book of reference, but its utility will be greatly enhanced if many inaccuracies are corrected. (For example, the external examiners at Birmingham and Bristol are put down as the Professors of Surgery.) The alphabetical list of consultants and specialists is an original feature and may serve a useful purpose.

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## *EPONYMS.*

BY SIR D'ARCY POWER, K.B.E., LONDON.

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### **XIX. THOMAS'S HIP SPLINT.**

HUGH OWEN THOMAS is a good example of a class of men who are not rare in the profession of medicine. Engaged in a busy general practice, Nature had endowed him with keen observation and with a mechanical genius which he turned to account in the treatment of his patients, untrammelled by any thought of the scientific or pathological principles involved. Nature to him was a personality working always towards a cure, and her efforts were best helped by rest. Thomas devoted all his energies to secure rest. In broken bones, in diseased joints, in obstructed bowels, he immobilized the parts, and by unrenitting attention to minute detail he assured himself that the rest was uninterrupted, for he took the absence of pain as his criterion.

He was born at Tyn Llan, Bodedern, in the Isle of Anglesey, on August 3, 1834, whilst his mother was on a visit to her parents, and was the eldest of five boys, all of whom became doctors. His father, Evan Thomas, was a bone-setter practising at 3 Great Crosshill Street, Wallasey, a suburb of Liverpool. He was the seventh in linear descent of a family believed by the neighbours to be endowed with a special gift of healing. Some of the members were well-to-do farmers who bestowed their gift without fee or reward like Cosmas and Damian our patron saints; the poorer members, like Evan Thomas, took money, but as a business it was not lucrative.

Hugh Owen Thomas, the son, was apprenticed to his maternal uncle, Dr. Owen Roberts, of St. Asaph's, at the age of 17, and as Dr. Roberts was surgeon to the workhouse and infirmary he was fortunately situated. He entered the University of Edinburgh at the age of 22, and had to maintain himself on the allowance of ten shillings a week, which was all that his father gave him. Here he came under the influence of Hughes Bennett, Professor of the Institutes of Medicine, who taught his students to observe precisely and methodically for themselves and to employ all modern methods of precision. Probably at the suggestion of the Rev. Thomas Guthrie, D.D., the great Edinburgh preacher of the day, he became secretary of a temperance

society and maintained its principles during the rest of his life. He left Edinburgh in 1856 without taking a degree, and entered as a medical student at University College in London. He obtained the diploma of M.R.C.S. Eng. in 1857, and immediately went to Paris, where he spent a few weeks and especially admired the skilful work of the French makers of surgical instruments. He then returned to Liverpool to help his father, but the rule-of-thumb methods adopted were une congenial to his better educated mind, and in 1859 he moved to a small house in Hardy Street which he occupied until 1870, when he moved to 11, Nelson Street. He soon gained a great reputation, first amongst the clubs and industrial societies to which he became attached as medical officer, and afterwards amongst the better classes in the city and its immediate neighbourhood. Money, though useful and necessary, was of secondary importance when weighed against the care of his patients, and he was idolized by the poor of Liverpool, whom he used to treat gratuitously every Sunday morning, although during the remainder of the week some fee was expected and taken. These poor attended his funeral literally in thousands, and showed genuine grief at the loss they had sustained.

A pale, thin man, 5 ft. 4 in. in height, always delicate, he was remarkable for the intelligence shown in his eyes, though the expression of his face was somewhat spoilt by an ectropion resulting from an accidental injury whilst he was a student at Edinburgh. He was an exceptionally keen observer, with a marked appreciation of symmetry or its absence, a trait inherited perhaps from his bone-setting ancestors. A man of the very simplest habits, he never took a holiday, and worked from six in the morning until midnight. His slight figure, closely buttoned in a blue coat, and wearing a naval cap because it had a peak which shaded an eye weakened by an ectropion, was familiar in all parts of Liverpool as he was driven about by a pair of black horses in his home-made gig. He smoked cigarettes almost continuously, and, being of an even temper, he was easy to live with. He was wholly without guile, and was ready to impart his knowledge and show his methods to everyone who sought him. Music was his chief hobby outside the workshop, and his delight was to play the flute whilst his wife accompanied him on the piano. He knew Lueretius almost by heart, and in later life he interested himself in archaeology, chiefly Egyptian. He was happily married, though childless. Of outside honours he received but one, the M.D. of the University of St. Louis, which was conferred upon him two years before his death.

Although Thomas is most generally known by the ingenious splints he invented, he was the author of several works which considerably influenced surgical opinions and methods. Amongst these were *Chronic Joint Disease; Compound Fracture of the Lower Jaw; Dislocations; Diseases of Joints; Fractures of the Upper and Lower Extremities; On Hip, Knee, and Ankle; Intestinal Obstruction; New Lithotomy Operation; Nerve Inhibition; Surgical Essays*; and many articles of a polemical character. His special surgical activities were directed to tuberculous arthritis. He preached the doctrine of prolonged and uninterrupted rest, and invented his splints for recumbency and also for early ambulatory exercise. Excision and amputation were almost the routine methods adopted by his contemporaries in the treatment of chronic disease of



the joints. His teaching did much to originate a more conservative treatment, and the methods he practised fifty years ago are those in common use to-day. He introduced many ingenious devices for the cure of fractures, and his patients rarely had more than half an inch of shortening after fracture of the femur. He was amongst the first to use percussion for ununited fractures, and he employed venous congestion long before it was used by Bier. He was, too, the first to insist upon the need for keeping paralysed muscles in a state of relaxation in cases of poliomyelitis. His versatility was shown in his treatment of intestinal obstruction at a time when no operation was



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performed for its relief. He often kept such patients starved for thirty or forty days, only allowing them a little arrowroot and water, giving hypodermic injections of morphia to check peristalsis and copious draughts of water to percolate through the inflamed intestinal area. He set his face rigidly against enemata. By these means he saved many lives at a time when it was usual to give castor oil and administer turpentine enemata to relieve the obstruction. The Museum of the Royal College of Surgeons of England contains a most interesting case of instruments which he invented for the operation of lithotomy, an operation in the performance of which he was very expert.

Although rough in manner, he gained his results, as well as the confidence of the patients and their friends, by his constant and personal attention to details and by the extreme gentleness of his manipulation. There could, indeed, be no greater proof of the confidence he inspired than his ability to keep a north countryman on a starvation diet for a month. The apparatus he employed was made at home and under his own eyes, and to this end he kept a saddler and a blacksmith in constant employment, whilst his workshop was fitted with the best lathes that money could buy. He died on January 6, 1891, and his practice was carried on by his nephew on the maternal side, my friend Sir Robert Jones, K.B.E., to whom I am indebted for many of the personal facts in this short notice.

The photograph of Mr. Thomas is from one kindly supplied by Sir Robert Jones.

*(To be continued.)*

## THE PATHOGENESIS AND TREATMENT OF TRAUMATIC NEURITIS OF THE ULNAR NERVE IN THE POST-CONDYLAR GROOVE.\*

BY HARRY PLATT, MANCHESTER.

### INTRODUCTION.

WHEN the ulnar nerve reaches the post-condylar groove at the elbow it comes to occupy a position of extreme vulnerability; but it is not the exposed situation of the nerve-trunk which alone determines the incidence of traumatic lesions at this level. Under normal conditions the nerve, though securely anchored in the groove, is also mobile enough to accommodate itself to the repeated changes in tension which occur during the movements of flexion and extension of the elbow-joint. This mechanism is readily disturbed when, as a result of injury, the nerve acquires a limited range of excursion or no longer conforms to the shape or orientation of its bed. In a certain small proportion of individuals the nerve is hypermobile, and, in the position of full flexion of the elbow, slips forward on to the epicondyle. This anatomical peculiarity may pass unnoticed for many years until a complete dislocation occurs, and the frequent repetition of the displacement gives rise to the symptoms of traumatic neuritis.

In the pathogenesis of certain types of ulnar-nerve injury an alteration in the normal relation between the nerve and its bed plays an all-important rôle. Such lesions do not ordinarily embrace the highly destructive primary injuries of this nerve which result from gunshot wounds or other traumata equally grave, although this factor may operate in some measure even in these conditions.

The special injuries which alone form the subject of this paper may be divided somewhat arbitrarily into three clinical groups: (1) *Ulnar-nerve lesions associated with recent fractures of the lower end of the humerus*; (2) *Late ulnar-nerve involvement after fractures*; and (3) *Recurring dislocation of the ulnar nerve*. In all three groups the actual nerve injuries belong for the most part to the class of incomplete lesions, and may thus be included under the broad title of traumatic neuritis.

### 1. ULNAR-NERVE LESIONS ASSOCIATED WITH RECENT FRACTURES OF THE LOWER END OF THE HUMERUS.

#### THE INCIDENCE OF THE NERVE LESIONS.

Considered as a whole, the ordinary fractures of civil life are but rarely complicated by the presence of injuries of peripheral nerves. In certain

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\* From the Orthopædic Service, Ancoats Hospital, Manchester.

regions, however, a combination of the two lesions provides a clinical picture which is by no means unfamiliar. The anatomical distribution of these dual injuries is shown in a recent paper by Dean Lewis and Miller,<sup>1</sup> of Chicago, who have collected 239 examples from surgical literature and from their own hospital records. The figures of these writers indicate that 80 per cent of the cases are fractures of the *humerus*, the common site of the fracture being in the lower third of this bone (60 per cent). Of the peripheral nerves implicated, the musculospiral accounts for 60 per cent, and the *ulnar nerve* for 25 per cent of the cases.

One of the earliest and most important investigations on this subject appeared in 1899 in the form of an admirable study by Broca and Mouchet,<sup>2</sup> of Paris, of the nerve lesions complicating certain fractures of the lower end of the humerus: 78 consecutive fractures of this type were observed during a period of two years, and in 9 of them a nerve injury was recorded. On close analysis these fractures were divided into two groups: (a) Fractures of the *external condyle*, of which there were 40 examples; and (b) *Supracondylar* fractures, in the remaining 38. In the former group there were 3 nerve injuries, and in the latter 6. In several cases the nerve lesions were multiple. The nerve-trunks involved were the ulnar (5 cases), median (5 cases), and the musculospiral (3 cases). It is interesting to note that during the same period in the practice of the authors there were no nerve injuries accompanying dislocations of the elbow-joint, other types of fracture of the lower end of the humerus, fractures of the olecranon, or fractures of the head of the radius.

In this paper of Broca and Mouchet the pathogenesis of the nerve injuries was considered in some detail. Special emphasis was laid on the chronology of the lesions, and a distinction made between *primary*, *secondary*, and *tardy* injuries, the latter term being applied to what had long been recognized as a clinical entity, viz., the late involvement of the ulnar nerve after fractures sustained in early childhood. Mouchet himself had already drawn attention to this delayed type of ulnar-nerve lesion in his Paris thesis submitted in 1898, and had realized that the injury which preceded it was almost invariably a fracture of the *external condyle*. Since that date, Mouchet has closely identified himself with this subject in a number of valuable contributions, and most notably in a comprehensive paper which appeared in the *Journal de Chirurgie* in 1914.<sup>3</sup>

The attention which has been focused in recent years on this interesting but relatively uncommon form of late ulnar-nerve lesion has tended perhaps to obscure the significance of the occasional involvement of this nerve in recent fractures of the elbow region. My own interest in this subject was first aroused a few years ago by the discovery, from time to time, of ulnar-nerve lesions amongst the recent elbow fractures treated in the fracture clinic of my surgical unit. Whilst it was obvious at once that the nerve injury was usually a transitory clinical phenomenon, in a few cases the interference with the conduction in the nerve was sufficiently grave to merit serious consideration. With a view to the acquisition of knowledge regarding the incidence and prognosis of these dual lesions in ordinary routine fracture treatment, a survey was made of the elbow fractures dealt with in this clinic over a four-year

period, viz., 1921 to the end of 1924. The number of such fractures under treatment was 329, and they were classified as follows :—\*

<i>Humerus</i> .—Supracondylar fractures	..	..	95
Fractures of the internal condyle	..	..	74
Fractures of the external condyle	..	..	83
<i>Olecranon</i>	..	..	38
<i>Radius</i> .—Head and neck	..	..	39
Total	..	..	329

In this series, 9 fractures (*Series I*)—all involving the *humerus*—were accompanied by a nerve injury, the nerve affected in each case being the *ulnar*. The absence of nerve lesions in the *olecranon* and *radius* fractures, and the immunity of the median and musculospiral nerves in this series, are facts of interest, but they do not demand special comment here.

*Eight* of the nerve lesions accompanied fractures of the *internal condyle*, and *one* a supracondylar fracture. The fractures were all of the 'closed' (simple) type. In *seven* cases spontaneous recovery was seen, full conductivity being restored in periods varying from three to eight months. In *one* case, however, at the end of nine months the clinical signs of a severe neuritis still persisted, and for this reason exploration of the nerve was undertaken. Following the operation of displacement of the nerve in front of the internal condyle rapid recovery ensued. The remaining patient, who presented the clinical signs of a very mild nerve lesion, ceased to attend the clinic after the first few weeks, but it is not unreasonable to assume that full recovery occurred.

If the group of fractures quoted here may be regarded as a typical consecutive series, it is evident that involvement of the ulnar nerve is an infrequent complication of fractures of the lower end of the humerus (9 in 252 fractures—roughly 3·7 per cent). A brief résumé of the clinical histories of these complicated injuries will now be presented before we embark on a more extended inquiry into the various factors which may be concerned in the pathogenesis of such nerve lesions.

## RECORDS OF CASES.

### SERIES I. CASES 1-9.

*Case 1.*—H. B., age 13 (1921). Fracture of internal condyle (fracture involving the joint surface).

ULNAR-NERVE INVOLVEMENT.—Signs of complete nerve-block noticed at the end of four weeks.

\* The classification adopted here for fractures of the lower end of the humerus is a simple one, which has proved convenient in the routine work of a fracture clinic. Each variety includes a number of subdivisions. Thus in the *supracondylar* group there will be a few uncommon or rare types, such as the T-shaped fracture and the diacondylar fracture. The *internal condyle* fractures include both fractures of the whole condyle and fractures limited to the epicondyle. Similarly, under the heading of *external condyle* fractures are included such rare injuries as isolated fracture of the capitellum.

It must also be explained that the fractures in the above series were received in the clinic direct from the casualty department of the hospital. The figures do not include complicated fractures treated elsewhere in the initial stage and referred for a consultation at a later date.

*Neurological Syndrome.*—Wasting with paralysis of the interossei, adductor pollicis, and hypothenar muscles; complete sensory loss. Traumatic ulceration on the dorsum of the little finger.

**SUBSEQUENT COURSE.**—

*Result of the Nerve Injury.*—Rapid spontaneous recovery. No sign of involvement three years later.

*Result of the Fracture.*—Full mobility of the elbow; no increase in the carrying angle. The internal condyle is thickened and the nerve is placed more superficially.

*Case 2.*—V. C., age 12 (1922). Fracture of internal condyle (fracture involving the joint surface).

**ULNAR-NERVE INVOLVEMENT.**—Signs of an incomplete nerve-block were noted at the end of three weeks.

*Neurological Syndrome.*—Flattening of the hypothenar eminence and interosseous spaces, with paresis in these muscles; analgesia in the ulnar area of the hand.

**SUBSEQUENT COURSE.**—

*Result of the Nerve Injury.*—Spontaneous recovery with almost complete restoration of function at the end of eight months. At the end of sixteen months no trace of any interference in the conductivity of this nerve.

*Result of the Fracture.*—Full range of flexion, but extension falls short of the full range by 10°. Moderate degree of cubitus valgus deformity.

*Case 3.*—A. B., age 12 (1922). Fracture of internal condyle (epicondyle only).

**ULNAR-NERVE INVOLVEMENT.**—Incomplete loss of conduction noted at the end of three weeks.

*Neurological Syndrome.*—Analgesia of the little finger, with paresis and flattening of the hypothenar muscles.

**SUBSEQUENT COURSE.**—

*Result of the Nerve Injury.*—Rapid spontaneous recovery, complete at the end of four months.

*Result of the Fracture.*—Full restoration of mobility of the elbow, with no increase in the carrying angle.

*Case 4.*—E. G., age 14 (1922). Fracture of internal condyle (fracture involving the joint surface).

**ULNAR-NERVE INVOLVEMENT.**—Signs of an incomplete block noted at the end of four weeks.

*Neurological Syndrome.*—Flattening and paresis of the hypothenar muscles, with full ulnar sensory loss.

**SUBSEQUENT COURSE.**—

*Result of the Nerve Injury.*—Spontaneous recovery with complete restoration of conduction at the end of eight months.

*Result of the Fracture.*—Full range of mobility of the elbow-joint with a normal carrying angle. The internal condyle is thickened and the ulnar nerve appears to be more superficial than on the opposite side, but is not unduly tender.

*Case 5.*—A. H., age 16 (1922). Fracture of the internal condyle (fracture involving the joint surface).

**ULNAR-NERVE INVOLVEMENT.**—Signs of an incomplete nerve-block noted at the end of two weeks.

*Neurological Syndrome.*—Analgesia in the ulnar area, but no appreciable interference with the motor power in the ulnar intrinsic muscles.

**SUBSEQUENT COURSE.**—The patient ceased to attend the clinic at the end of six weeks; at that time the elbow-joint had regained full mobility. It is presumed that the nerve lesion recovered completely. Repeated efforts to trace the patient have been unsuccessful.

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*Case 6.*—F. B., age 12 (1922). Fracture of internal condyle (epicondyle only).

ULNAR-NERVE INVOLVEMENT.—Signs of an incomplete nerve-block noted at the end of three weeks.

*Neurological Syndrome.*—Analgesia in the ulnar area, with paresis and wasting of the hypothenar and interossei muscles.

SUBSEQUENT COURSE.—

*Result of the Nerve Injury.*—Spontaneous recovery with complete restoration of conduction at the end of six months.

*Result of the Fracture.*—Full range of mobility at the elbow with no alteration in the carrying angle.

*Case 7.*—W. H., age 12 (1922). Fracture of internal condyle (fracture involving the joint surface).

ULNAR-NERVE INVOLVEMENT.—Signs of an incomplete nerve-block noted at the end of three weeks.

*Neurological Syndrome.*—Marked paræsthesia, with an incomplete sensory loss; wasting and paresis of the interossei and hypothenar muscles.

SUBSEQUENT COURSE.—

*Result of the Nerve Injury.*—Condition nine months later: All the signs of a severe ulnar neuritis. The nerve-trunk is exquisitely tender on palpation, and in full flexion of the elbow is abnormally superficial. There is complete paralysis of all the ulnar intrinsic muscles, with marked tenderness of the hypothenar eminence. Moderate clawing of the little and ring fingers is present, and there is an incomplete sensory loss. Operative exploration of the nerve considered advisable.

*Result of the Fracture.*—The elbow at this date allows the full range of flexion, and practically a complete range of extension. There is a definite increase in the carrying angle.

OPERATION.—Neurolysis with anterior transposition. The nerve in the region of the groove showed a restricted mobility, and was slightly thickened, the area of the lesion being represented by an early fusiform neuroma. The nerve was freed, displaced in front of the internal condyle, and buried in an intramuscular bed.

Rapid subsidence of the signs of irritation, with steady return of conduction.

Condition eighteen months after the operation: Complete restoration of conduction in the ulnar nerve, with no appreciable impairment of function.

*Case 8.*—C. S., age 8 (1924). Fracture of internal condyle, complicating a posterior displacement of the elbow-joint.

After reduction of the dislocation, the elbow was rested in the position of full flexion for two weeks. Active movements and massage followed.

ULNAR-NERVE INVOLVEMENT.—Signs of incomplete loss of conduction noted at the end of six weeks.

*Neurological Syndrome.*—Very slight flattening of the muscles bellies of the flexor carpi ulnaris, hypothenar eminence, and interossei. No appreciable paresis; no trace of sensory loss. The ulnar nerve at the elbow is slightly more superficial than on the opposite side.

SUBSEQUENT COURSE.—

*Result of the Nerve Injury.*—Complete recovery of condition and function eight months later.

*Result of the Fracture-dislocation.*—Full range of mobility in the elbow-joint was restored. The internal condyle remains thickened and irregular.

*Case 9.*—J. P., age 5 (1923). Supracondylar fracture.

Considerable swelling in the early stages; backward displacement of the lower end of the diaphysis. Further readjustment of the fracture was necessary two weeks after the injury, and this was carried out under an anæsthetic.

ULNAR-NERVE INVOLVEMENT.—Signs of an incomplete interruption of conduction in the ulnar nerve were noted at the end of six weeks.

*Neurological Syndrome.*—Wasting and paralysis of the hypothenar muscles and hypesthesia in the ulnar skin area; slight tendency to clawing of the little finger;

marked tenderness of the hypothenar muscels. Nerve-trunk at elbow definitely tender, but not palpably thickened.

SUBSEQUENT COURSE.—

*Result of the Nerve Injury.*—Spontaneous recovery with practically complete restoration of function at the end of eight months.

*Result of the Fracture.*—Full mobility of the elbow-joint, with a barely appreciable increase in the carrying angle.

To the above may now be added, for comparative purposes, three further examples (*Series II*) of severe ulnar-nerve lesions complicating (a) an internal condyle fracture, and (b) two cases of posterior dislocation of the elbow-joint. These patients had received treatment in the early stages elsewhere, and came under observation after several weeks had elapsed from the date of the injury. In two cases it was considered advisable to explore the injured nerve.

SERIES II. CASES 10-12.

*Case 10.*—T. C., age 20 (1924). Fracture of internal condyle, complicated by a small chip fracture of the coronoid process of the ulna.

Patient was seen after eight weeks' treatment elsewhere. The elbow had been fixed in full flexion for ten days, and at the end of that period *energetic passive movements* were begun. *Involvement of the ulnar nerve* was recognized about the end of the fourth week.

CONDITION WHEN FIRST SEEN.—The elbow showed considerable distortion in the region of the internal condyle, and there was marked limitation of the range of mobility of the joint. There was an obvious ulnar-nerve lesion, with all the signs of a complete block. In view of the syndrome, and the experience of previous cases, exploration of the nerve-trunk was considered advisable.

OPERATION.—The ulnar nerve was exposed from the lower third of the upper arm to the junction of the upper and middle thirds of the forearm. There was considerable scarring of the aponeurosis and muscle-fibres of the common flexor origin from the internal condyle. The nerve-trunk was intact, but in the region of the ulnar groove was extremely attenuated and reduced to a narrow flat ribbon over a length of one inch. The area of the lesion was demarcated above and below by a small neuroma involving the whole thickness of the nerve-trunk. Complete resection of the area of the lesion was carried out, and *end-to-end suture* performed, the sutured nerve being *displaced* in front of the internal condyle and embedded in the usual way.

*Case 11.*—C. M., age 15 (1924). Posterior dislocation of elbow-joint.

This patient also was treated elsewhere in the early stages. After reduction of the dislocation, the elbow had been rested for ten days and *forcible passive movements* begun at the end of that time. The ulnar-nerve lesion was recognized about the end of the fifth week.

CONDITION WHEN FIRST SEEN (thirteen weeks subsequent to the injury).—The elbow-joint showed practically an undiminished range. There was an ulnar-nerve involvement, with all the signs of a severe but incomplete block. Exploration of the nerve-trunk advocated.

OPERATION.—The ulnar nerve was exposed in the lower third of the upper arm as far as the junction of the upper and middle thirds of the forearm. In the whole area exposed the nerve-trunk was found to be anchored to its bed by thin filmy strands, so that its mobility was very considerably restricted. This abnormal anchorage was best marked in the groove. The sheath of the nerve showed many points of vascular injection, and in the region of the groove the trunk was reduced to a calibre representing about one-half of the normal cross-section. There was no definite bulb, but there was an appreciable amount of induration at the upper limit of the attenuated segment. It was noted also that the fibres of origin of the superficial flexor muscles from the internal condyle contained a good deal of tough scar



tissue. The nerve-trunk was *displaced* in front of the internal condyle and buried in the usual fashion.

*Case 12.*—J. D., age 16 (1920). Posterior dislocation of elbow-joint.

The patient was seen three weeks after the injury. Two unsuccessful efforts had already been made to reduce the dislocation, and *repeated forced movements* had been utilized in an attempt to reproduce mobility.

ON EXAMINATION.—The left elbow showed an extensive swelling and was completely fixed in the position of right-angle flexion. Palpation of the bony points suggested that the dislocation still existed. This view was confirmed by radiograms. All the signs of a *complete ulnar-nerve block* were present.

A further attempt was made to reduce the displacement by manipulation alone, but this failed, and accordingly an open operation was performed. It was found necessary to excise the lower end of the humerus. The ulnar nerve was not exposed at the time.

## SUBSEQUENT COURSE.—

*Result of the Nerve Injury.*—Steady spontaneous recovery was seen, and for this reason the nerve-trunk was not explored. Four years from the time of the injury no clinical signs of ulnar-nerve involvement could be demonstrated.

*Result of the Dislocation* (excision of the elbow).—A distorted elbow, allowing full flexion and almost complete extension, with adequate stability.

## PATHOGENESIS.

It is desirable first of all to define the chronological relation between the fracture and the nerve lesion, for if the nerve injury is produced at the same moment as the fracture—a primary lesion—the incidence of this complication is beyond the control of the surgeon. But if it is clearly proved that the clinical signs of involvement of the nerve-trunk have arisen *de novo* in the first few weeks after the injury—a *secondary* lesion—the methods employed in the early treatment of the fracture must be subjected to critical scrutiny.

Secondary nerve lesions are usually regarded as due to the compression effect of callus or to the continued pressure of an unreduced bony fragment. If by callus-compression is meant the actual incorporation of a nerve-trunk in newly formed fibro-osseous tissue, then in my opinion this is an extremely rare cause of nerve injury associated with fractures. In more than 600 personal operations on injured nerves, complete ensheathment of a nerve in callus has been seen twice only. But exuberant callus formation may easily produce a distortion of the nerve-bed, and so place the nerve under conditions of abnormal tension or friction. Under such circumstances a traumatic neuritis may be excited by the ordinary movements of the neighbouring joint. The kinking, or the gradual laceration of a nerve-trunk over a bony projection, is a well-recognized cause of nerve injury, and one which has been demonstrated on many occasions during exploratory operations.

In the treatment of fractures of the elbow region both these potential etiological factors are almost entirely under the control of the surgeon. Thus it is common knowledge that excessive callus formation is almost invariably the result of the injudicious use of forced movements of the elbow-joint in the early stages. It is also obvious that, with an early and efficient reduction of any bony displacement, the risk of a secondary nerve injury due to this cause is negligible. But even when such definite gross therapeutic errors are

excluded, it is clear that there are occasional fractures in which a secondary nerve lesion must be accepted as an inevitable complication.

The chronological definition of any given nerve injury must often be based on careful clinical observation. It is generally taught that the syndromes of a primary and a secondary nerve lesion present certain differences. In the former the onset of the signs of nerve-block is abrupt, and the loss of conduction rapidly becomes complete. In the latter the onset is more gradual, and the signs of block deepen slowly and may remain incomplete for an indefinite period. But in actual practice these distinctions are not always clear-cut, and from clinical records alone it is by no means easy to assign a nerve injury to its appropriate chronological group. This difficulty is illustrated when we attempt to classify the individual cases in the first series quoted above (*Cases 1-9*). It is seen here that in no case was the syndrome of a nerve lesion identified before the third or fourth week; and further, in most of the patients the signs of an incomplete nerve-block only were present. At first sight these facts would appear to support the view that the lesions were all of the secondary type. In this assumption, however, there lies a possible fallacy, for during the first two or three weeks the local signs of bony trauma dominate the clinical picture, and a peripheral nerve lesion may easily escape notice, even though that lesion dates from the time of the injury. This applies exclusively to incomplete lesions of the *ulnar* and *median* nerves; the wrist-drop of the *musculospiral* palsy, on the other hand, is rarely missed at the onset.

The nerve injuries in the first series of cases, in my judgement, belong to the *primary* class. In the *internal condyle* fractures this view seems incontestable when the mechanism of the production of the fracture is reviewed, for it is evident that the nerve sustains a direct contusion at the moment the condyle is chipped off. Moreover, as the separated condyle is usually drawn forwards away from the nerve by the pull of the flexor muscles, the local conditions do not ordinarily favour the production of a secondary lesion if the early treatment is on correct lines. *Case 9*, a *supracondylar* fracture, is perhaps more difficult to place, but may not unreasonably be classified as a primary lesion.

In the second series similar difficulties arise, but on the whole it seems fair to allocate these lesions also to the primary class.

This decision, however, does not warrant the assumption that either the severity of the lesion or the fate of the injured nerve is determined alone by the primary trauma. In the type of nerve injury under consideration, the distinction between primary and secondary lesions is really of little moment. For it is quite clear that during the early stages of treatment a mild primary contusion may be converted into a severe progressive neuritis. A primary lesion may then pass into the secondary class. This sequence of events is well illustrated in certain cases in both series recorded above.

In *Case 7* of the first series—the one example of a severe and persistent neuritis—the early treatment may justifiably be absolved from any direct share in the production of the nerve lesion. The elbow was treated from the beginning in accordance with the routine for all such fractures, viz., rest in full flexion for a period of at least two weeks, followed by very gradual

mobilization of the joint by means of *active* movements only. When the signs of ulnar-nerve irritation became apparent, efforts to reproduce joint mobility were suspended. In spite of this, the intensity of the neuritis, as measured both by subjective and objective signs, deepened steadily. Here was a severe primary contusion of the nerve associated with a slight disturbance of the nerve-bed due to the fracture. In consequence the swollen nerve became more superficially placed, and its range of excursion in the groove was curtailed. In addition, at the end of a few months the elbow showed a mild cubitus valgus deformity and the full range of extension could not be obtained. In short, a combination of factors capable of producing a friction or tension neuritis in the anchored nerve. The secondary neuritis in this case, in my opinion, was an unavoidable complication. The dramatic effect of the operation of anterior transposition of the nerve adds further testimony to this conception of the pathogenesis of the nerve injury.

In the remaining cases, which were all treated in the routine fashion, the factors described above did not intervene, and the nerve lesions recovered speedily.

In the second series we find three primary nerve lesions in which serious additional damage was inflicted in the first few weeks by injudicious treatment, i.e., the use of repeated *forced passive* movements. The clinical picture in these cases again illustrates the evolution of a friction or tension neuritis; but here the gravity of the lesions was determined by therapeutic mistakes.

It is important to recognize that in all the cases recorded above the harmful effect of forced movements was not dependent on excessive callus formation. Indeed, where exuberant callus and a nerve lesion co-exist, both complications may be produced by a common etiological factor.

#### SUMMARY.

1. Involvement of the ulnar nerve is a rare complication in recent fractures of the lower end of the humerus (9 in 252 consecutive cases in the writer's series). The common type of fracture to be accompanied by a lesion of this nerve is separation of the *internal condyle*.

2. The nerve lesion is usually a primary contusion—a physiological block without loss of continuity. The symptoms are slight, and rapid spontaneous recovery tends to occur.

3. In exceptional cases the *primary* neuritis may increase in severity during the early stages of treatment, and the lesion may pass etiologically into the *secondary* class.

4. A *secondary* ulnar neuritis of this type may be an unavoidable sequela. In such cases a friction or tension neuritis is produced as a result of a disturbance of the normal relation between the nerve and its bed.

5. A *secondary* neuritis may also be induced by injudicious treatment, i.e., the use of forced passive movements of the elbow in the first few weeks after the injury. Under similar circumstances a secondary neuritis may arise *de novo*.

6. In all cases of severe and persistent neuritis the nerve-trunk should be explored and displaced to a new bed in front of the internal condyle.

## 2. LATE ULNAR-NERVE INVOLVEMENT AFTER FRACTURES.

It is believed that Duchenne was familiar with this condition, but as far as can be ascertained it was first described as a clinical entity by Panas<sup>4</sup> in 1878. Prior to 1900, cases were recorded by Bowlby, Weber, Guillemin and Mailly, and others; and to this period belongs the first of Mouchet's sequence of illuminating papers. Since that time the subject has received considerable attention from both surgeons and neurologists. Prominent in the more recent literature are the contributions of Sherren,<sup>5</sup> Ramsay Hunt,<sup>6</sup> Adson<sup>7</sup> of the Mayo Clinic, Buzzard,<sup>8</sup> Lusena,<sup>9</sup> Dean Lewis and Miller (*loc. cit.*)\*

The broad clinical picture, which is a characteristic one, has long been recognized. In an adult who has sustained a fracture of the lower end of the humerus in early childhood, the subjective and objective signs of neuritis of the ulnar nerve arise insidiously. The neuritis is slowly progressive, and after a time the nerve in the region of the elbow becomes thickened, and may show a distinct fusiform neuroma (nerve spindle). Many of the earlier cases were regarded as examples of compression neuritis due to the involvement of the nerve in old 'callus', and this idea, though early shown by Mouchet to be illogical, dominated the operative treatment of the lesion for many years. Various types of humerus fracture have been described as antedating this late form of nerve lesion. In 43 cases collected by Dean Lewis and Miller, the primary bony injury was as follows: external condyle fractures, 23; internal condyle fractures, 10; supracondylar fractures, 8; 'epiphyseal separation', 2. Late ulnar palsy has also been described in association with an old infective arthritis of the elbow-joint (Weber, Sherren).

It is important to remember, however, that many of the earlier cases were recorded either before the advent of radiography or at a time when radiographic technique was admittedly imperfect. For this reason the existing descriptions of the bony injury are often unreliable; in a number the type of fracture has been rather a matter of clinical conjecture. This, as we shall see, is a consideration of some importance in connection with the pathogenesis of the nerve lesion. Mouchet's original case followed a fracture of the *external condyle*, and by 1914 he had dealt with seven cases, all corresponding to this one clinical type. (Since 1914 he has seen three additional cases.) In his view, the classical late ulnar-nerve lesion is unlikely to be associated with the other varieties of humerus fracture, because the development of the neuritis is determined primarily by the existence of a gross cubitus valgus deformity. Now this deformation—in an extreme degree—is a characteristic sequela of the complete external condylar fracture and no other, although an inconspicuous increase in the carrying angle is not infrequently present after supracondylar or even internal condyle fractures. It may be said at once that a scrutiny of the cases reported in the last decade, accompanied by full radiographic evidence, bears out the accuracy of Mouchet's conception, i.e., that the late ulnar-nerve lesion is ordinarily a sequela of fractures of the external condyle. This does not preclude the possibility of late involvement occurring after other types of humerus fracture, but such cases are quite exceptional.

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\* Since the greater part of this paper was written some two years ago, further important contributions have been made both by Mouchet<sup>10</sup> and Miller.<sup>11</sup>

With this brief introduction we may now proceed to consider in greater detail the clinical features, pathogenesis, and surgical treatment of late ulnar palsy. It will be convenient at this stage to interpolate the clinical records of a series of personal cases (*Series III*) which will serve to illustrate and emphasize many points in the description which follows.

## RECORDS OF CASES.

### SERIES III. CASES 13-18.

*Case 13.*—Miss R., age 59. Old fracture of external condyle of left humerus. Late involvement of ulnar nerve.

**HISTORY.**—At the age of 5 sustained a fracture of the lower end of the humerus which left a deformity of the elbow and some limitation of mobility. *Fifty-one years after* the injury, began to experience pain and tingling in the little and ring fingers. Two years after the onset of these symptoms the patient sustained a contusion of the elbow, and from this time onwards her condition became steadily worse. The pain was evoked during the movements of flexion of the elbow and supination of the forearm.

**CONDITION ON EXAMINATION.**—The left elbow showed a marked cubitus valgus deformity with irregularity of both condyles. Movements of the joint were restricted, extension falling short by about one-third of the full range, but flexion was almost complete. At the elbow the ulnar nerve was unduly palpable, and there was an appreciable degree of thickening of the trunk, which was also exquisitely tender.

#### *Neurological Syndrome.*—

*a.* Motor function: The signs of an incomplete nerve-block were present. There was paresis and wasting in the belly of the flexor carpi ulnaris and inner half of the flexor profundus digitorum; but the ulnar intrinsic muscles of the hand were all acting, and there was no visible atrophy.

*b.* Sensory function: Definite hypoesthesia in the ulnar area of the hand, but the protopathic response was normal. There were no vasomotor paralysis or trophic changes.

*A radiogram (Fig. 270) showed an old fracture of the external condyle of the humerus with non-union.*

**OPERATION.**—Feb. 20, 1921. *Exploration of the ulnar nerve* in the region of the elbow, with *anterior displacement*. In the region of the post-condylar groove the nerve-trunk was found to be slightly swollen, and where lying in contact with the joint capsule a tiny opening was seen which led into the joint cavity. The



**FIG. 270.**—*Case 13.* Radiogram of elbow. Late ulnar palsy. Shows: (a) The old fracture of the external condyle, with no sign of bony continuity fifty-three years later; (b) The apparent overgrowth of the internal condyle; (c) The cubitus valgus deformity.

cartilaginous covering of the humerus at this spot showed some irregularity, producing a nodular surface. On the deeper aspect of the nerve at this level the sheath was frayed through at one spot; elsewhere the nerve-sheath was thickened, and there appeared to be a slight degree of intraneural induration.

The nerve-trunk was freed in the lower part of the upper arm and upper part of the forearm, and was then displaced in front of the internal condyle, the proximal branches being preserved intact. The nerve was buried in an intramuseular bed.

**SUBSEQUENT COURSE.**—The operation was followed within a few weeks by complete cessation of the pain experienced during movements of the elbow. Three years later, all the subjective sensory symptoms had completely disappeared, except that the patient was still conscious of a slight numbness in the ulnar distribution of the hand. A full neurological examination at this date was not available.

**Case 14.**—Miss N., age 27. Old fracture of external condyle of left humerus. Late involvement of ulnar nerve.

**HISTORY.**—At the age of 3 sustained a fracture of the lower end of the humerus. As a result, the elbow-joint had always been deformed and there had never been a complete range of movement. The patient, who was now a professional violinist, during the past few months had become conscious of the development of progressive weakness in the left hand, and had experienced attacks of pain referred along the little finger. She had also noticed a certain amount of wasting of the finer muscles of the hand. In consequence she found increasing difficulty in continuing to play her instrument. (Onset of symptoms twenty-four years after the injury.)

**CONDITION ON EXAMINATION.**—The left elbow showed a marked deformation with a great increase in the carrying angle, and an undue projection or apparent overgrowth of the internal condyle. There was a full range of flexion, but extension fell short by about 20°. The ulnar nerve was easily palpable in the postcondylar groove, but did not feel enlarged. It was somewhat tender on compression, but this sign was not conspicuous.

**Neurological Syndrome.**—

*a.* Motor function: In the hand there was definite atrophy of the interossei, adductores pollicis, and hypothenar eminence. There was marked weakness in the adductor pollicis, interossei, and abductor minimi digiti; the flexor brevis pollicis showed a complete palsy.

*b.* Sensory function: There was diminished acuity of sensation in the ulnar area, both to light touch and painful stimuli.

A radiogram showed an old fracture of the *external condyle* with fibrous union.

**OPERATION.**—Nov. 4, 1922. Exploration of ulnar nerve; anterior transposition. In the region of the post-condylar groove the nerve was very slightly swollen and showed an appreciable degree of intraneural induration. The nerve-trunk was freed in the usual way, displaced forwards, and buried in an intramuseular bed.

**SUBSEQUENT COURSE.**—Within a few weeks after the operation definite and progressive signs of regeneration set in. The patient resumed her violin playing four months later, and eleven months from the time of the operation was doing full duty in an orchestra.

**Condition Two Years after the Operation.**—There was no trace of wasting of the ulnar intrinsic muscles, and sensory tests showed that there was a mere trace of hypo-aesthesia over the terminal phalanx of the little finger.

**Case 15.**—Miss D., age 33. Old fracture of external condyle of humerus. Late involvement of ulnar nerve.

**HISTORY.**—At the age of 2 sustained a fracture of the lower end of the left humerus. Perfect movement was restored in the joint, but in later childhood the elbow was noticed to be deformed. Two years ago (thirty-one years after the injury) the patient became conscious of an increasing loss of power in the left hand, particularly marked during piano playing, and also when handling a golf club. Later she began to have attacks of tingling along the inner border of the hand and little finger, and more recently felt that this region was numb. She had found that relief from the subjective symptoms could be obtained by pressing on the ulnar nerve at the elbow.

## TRAUMATIC NEURITIS OF ULNAR NERVE 421

**CONDITION ON FIRST EXAMINATION.**—The elbow showed a marked cubitus valgus deformity, the carrying angle being increased by about  $30^{\circ}$  beyond the normal. The region of the *internal condyle* was unduly prominent, and the whole of the lower end of the humerus appeared to be broadened. The normal relation of the bony points was distorted owing to the obliquity of the lower articular surface of the humerus. The post-condylar groove was shallow, and the ulnar nerve more palpable than usual. The nerve-trunk was obviously thickened, with the formation of a very slight 'spindle'; it was exquisitely tender, and on palpation there was marked tingling referred along the ulnar border of the hand.

*Radiograms* (Fig. 271) showed an old fracture of the external condyle.



FIG. 271.—Case 15. Radiograms of elbow, anteroposterior and semilateral views. Late ulnar palsy. Shows: (a) The old fracture of the external condyle; (b) The distorted internal condyle; and (c) The cubitus valgus deformity.

### *Neurological Syndrome.*—

*a. Motor function:* Marked atrophy in all the ulnar intrinsic muscles of the hand, with complete loss of voluntary power. The flexor carpi ulnaris and flexor profundus digitorum (inner half) were acting, but were definitely weak.

*b. Sensory function:* There was definite hypo-aesthesia and incomplete loss of protopathic sensation in the ulnar area of the hand. The deep sensation was normal. There were no conspicuous vasomotor changes, and no trophic signs.

**OPERATION.**—March 21, 1924. Anterior transposition of the ulnar nerve. Nerve-trunk in the groove was slightly thickened; sheath intact.

**SUBSEQUENT COURSE.**—Complete disappearance of the paresthesia signs two months after the operation.

*Case 16.*—Mrs. B., age 32. Old fracture of left external condyle. Late involvement of ulnar nerve.

**HISTORY.**—At the age of 6 sustained a fracture of the left elbow; a well-marked deformity had been present since the year of the injury, and the elbow-joint had never recovered full mobility. Four years ago (twenty-two years after the accident) the patient began to have attacks of pain radiating from the inner side of the elbow to the little finger. She had worked for many years in a cotton mill, duties which involved constantly repeated flexion and extension of the elbow. Shortly after the onset of pain, wasting of the small muscles of the left hand became apparent. At the time of examination, although still engaged in her occupation, the patient had begun to find it almost impossible to continue at work owing to the severe pain and the progressive weakness in the hand.



FIG. 272.—*Case 16.* Radiogram of elbow. Late ulnar palsy. Shows: (a) The old fracture of the external condyle with non-union; and (b) The cubitus valgus deformity.

**CONDITION ON FIRST EXAMINATION.**—The left elbow presented a conspicuous cubitus valgus deformity, with prominence and apparent over-growth of the internal condylar region. The external condyle showed an irregular projection, and the line of the articular surface of the humerus was oblique. The range of flexion was normal, but extension was restricted to some three-fourths of the full range. The ulnar nerve at the elbow seemed to be placed more superficially. It was freely mobile, but did not dislocate. The post-condylar groove was definitely shallow, and the interval between the olecranon and internal condyle appeared to be diminished as compared with the opposite side.

*Neurological Syndrome.*—

*a. Motor function:* The ulnar side of the forearm showed distinct atrophy in the region of the muscle bellies of the flexor carpi ulnaris and profundus digitorum, and the ulnar intrinsic muscles of the hand showed a more

extreme degree of atrophy. All these muscles were markedly parietic but were still acting. There was no claw deformity.

*b. Sensory function:* There was a definite hypo-aesthesia to light touch, with an exaggeration of the protopathic response. There were no vasomotor changes or trophic phenomena.

A radiogram (Fig. 272) shows a typical fracture of the external condyle, with fibrous union.

**OPERATION.**—Aug. 15, 1924. Exploration of ulnar nerve; anterior transposition. The operation was carried out with the usual technique. The following special points were noted: (1) The nerve-trunk in the groove was swollen, injected, and



the sheath very thin. There was no palpable induration. Below the level of the oedematous spindle the nerve was wasted and pale. Faradic stimulation above and below the area of the lesion gave a response in the flexor profundus digitorum, but none in the flexor carpi ulnaris or in the ulnar intrinsic muscles. (2) The muscle belly of the flexor carpi ulnaris as displayed at the operation was much attenuated, and consisted practically of a sheet of connective tissue.

**SUBSEQUENT COURSE.**—Rapid disappearance of the subjective symptoms occurred in the first few weeks after the operation.

**Condition Ten Months after the Operation.**—The atrophy of the ulnar intrinsic muscles is less marked, and there are signs of increase in power in this group. There is an absence of any sensory loss or vasomotor change. The nerve-trunk is evidently well embedded in front of the elbow, and there is no local tenderness in this region.

**Cases 17 and 18.**—These are two examples of late ulnar-nerve palsy in patients who came under my observation after operative treatment had already been carried out. In both, a considerable amount of bone had been removed from the lower end of the humerus in an attempt to reconstruct a new bony bed for the nerve-trunk. The original bone injury had obviously been a fracture of the external condyle. In each case the result of the operation had been unfortunate, as the clinical signs of a complete nerve-block had developed. Further exploration of the nerve-trunk was therefore considered advisable, and in both cases a large irregular indurated nerve spindle was found embedded in a mass of scar tissue in the region of the old post-condylar groove. The lesions demanded *resection* and end-to-end suture.

These cases are included in the series as they emphasize the dangers of the type of operative treatment which had previously been employed.

### CLINICAL FEATURES.

Late ulnar palsy has been seen more frequently in the male sex, as one would expect in a traumatic lesion of this type. It is convenient to recognize three clinical stages which form a definite sequence.

**1. The Fracture.**—The initial injury is sustained in early life, and usually in childhood between the ages of 2 and 10. In the typical case the fracture involves the external condyle of the humerus, the line of cleavage running obliquely into the elbow-joint. This is a familiar fracture in childhood, and one which often gives rise to difficulty in treatment owing to the tendency of the large fragment to be 'turned turtle' by the pull of the extensor attachments. It appears to be the rule for this fracture to unite by fibrous tissue only (Fig. 273). In many cases the functional result of the fracture as regards joint mobility is fairly satisfactory. The *cubitus valgus* deformity usually manifests itself at a comparatively early stage, but often excites little comment at the time. In adult life the distortion is always conspicuous. This is due in the early stage to the initial displacement of the condyle; later the effects of irregular epiphyseal growth are superadded.

**2. The Latent Period.**—In 75 per cent of the recorded cases the interval between the injury and the onset of the first signs of nerve involvement is not less than ten years. Latent periods of thirty years, or even more, are by no means rare (e.g., Case 13). During this time no change in the elbow is noted other than the steady increase in the degree of the deformity, which reaches its maximum with the cessation of growth.

### 3. Stage of Neuritis.

**a. Symptoms.**—Signs of interference with the function of the ulnar nerve arise insidiously and usually progress steadily. The neurological syndrome

of the initial phase is found to vary in different individuals. Thus the early subjective symptoms may be predominantly sensory or motor. If the former, the patient experiences local pain in the region of the elbow excited by movements of the joint, together with tingling and a sense of numbness referred to the ulnar area of the hand. When the motor signs take precedence, there is a slowly progressing weakness in the hand, and certain of the finer movements become increasingly difficult to carry out (*Case 14*).

The objective signs at first indicate the presence of an incomplete nerve-block, which as time goes on gradually deepens towards the stage of complete interruption. A partial sensory loss may be present, but it is often overshadowed by the irritative phenomena. The paresis and wasting of the intrinsic muscles of the hand supplied by this nerve soon become manifest. In long-standing and untreated cases a complete ulnar palsy with all its mechanical sequelæ may ultimately be seen. The trophic changes characteristic of an irritative nerve lesion are present in greater or lesser degree, and the patient runs the usual risk of local complications dependent on the existence of an analgesic area of skin.

*b. The Nerve-trunk.*—On palpation of the nerve in the post-condylar groove the local lesion is at once demonstrable. At first the nerve is exquisitely tender and slightly thickened; later the local induration is evident in the form of a painful nerve 'spindle'.

*c. Configuration of the Elbow.*—Reference has already been made to the cubitus valgus deformity which is always a striking feature. The distortion of the lower end of the humerus gives a false impression of overgrowth of the internal condylar



FIG. 273.—Typical fracture of the external condyle in a boy of 12. No bony union seven years after fracture; marked cubitus valgus deformity. No nerve involvement.

region. It is this appearance which has often been responsible for the erroneous clinical diagnosis of old fracture of the *internal condyle*. The ulnar groove is found to be relatively shallow, and in it the tender thickened nerve-trunk appears to lie in an unusually exposed and superficial position.

A radiogram of the elbow demonstrates the old fracture of the *external condyle*, invariably with no sign of complete bony union even at this date (see Figs. 270, 271, 272).

## PATHOGENESIS OF THE NERVE INJURY.

Mouchet was the first to give a logical explanation of the manner in which the nerve injury is produced. He pointed out that, as a result of the elbow deformity, the nerve is compelled to take a longer course, and tends to be stretched like a bowstring along the inner border of the olecranon. This stretching, however, does not occur during the movement of extension, as suggested originally by Mouchet, for in this position the nerve-trunk is relaxed. On the contrary, the abnormal tension comes into play during each movement of flexion, and the constant repetition of strong flexion movements provides a recurring trauma. It is easy to visualize the gradual development of a 'friction' neuritis under these circumstances, and the lesion illustrates this train of events most beautifully. It is difficult, however, to explain why the onset of the neuritis should be delayed for such a long latent period. From a study of clinical records it appears that the onset of nerve symptoms frequently coincides with a period of strenuous over-activity in which oft-repeated forcible flexion of the elbow is demanded. Such conditions may be provided by the special activities of sport or occupation (*see Cases 14, 15, 16*).

## TREATMENT.

There seems to be little scope for conservative non-operative methods in the treatment of delayed ulnar neuritis. Operative exploration has been practised in these cases for many years, but some of the procedures employed have a purely historical interest. Mouchet in his classical article published in 1914 selected four types of operation as being worthy of discussion at that date: (1) *Simple freeing of the nerve (neurolysis)*; (2) *Gouging out the postcondylar groove*; (3) *Supracondylar osteotomy*; (4) *Anterior transposition of the nerve (displacement-neurolysis)*.

1. **Simple Freeing.**—This operation was carried out by Potherat in 1897 on Mouchet's first case, but with no amelioration of the neuritic symptoms. In the light of our present knowledge concerning the pathogenesis of the lesion, the operation as a neurolysis is obviously inadequate, and may therefore be dismissed without further comment.

2. **Gouging out the Post-condylar Groove.**—For many years this operation has figured in the literature as a reputable method of dealing with the lesion under consideration. It was practised by Broca in 1899 with disappointing results, but occasional successes have been recorded, e.g., Sherren (1908).

The operation is open to criticism on *a priori* grounds, as it does not fulfil the canons of a properly designed neurolysis. It is clearly unwise to replace a nerve-trunk in a bed where its mobility is likely to be further restricted by the production of a fibro-osseous scar. Moreover, its course is not shortened to an appreciable degree. With the operations at our disposal now to be considered, efforts to enlarge the postcondylar groove cease to be of any practical value. The results of this operation in *Cases 17* and *18* in my series illustrate its potential dangers.

3. **Supracondylar Osteotomy.**—This is the procedure recommended by Mouchet and practised with excellent results in three cases in his original series. It involves the removal of a wedge, with its base on the inner side, from

the lower end of the humerus, thus correcting the cubitus valgus and at the same time shortening the course of the overstretched nerve-trunk. There is little to criticize in this operation, but it does not seem to have been adopted widely.

4. **Anterior Transposition of the Nerve.**—There are records of this operation having been carried out as long ago as 1898 by an American surgeon, with the report that the pain complained of was relieved. Roux, of Lausanne, operated by this method on one of the early patients seen by Broca and Mouchet, and to him is generally ascribed the introduction of this procedure. In this particular patient, examined some years later by Mouchet, the result was said to be disappointing.

Mouchet's criticism of the operation, as performed at that time, was that the nerve-trunk was left exposed in a subcutaneous position as it passed along its new course in front of the internal condyle. But this technique is no longer followed, for in recent years many opportunities of practising the operation of anterior displacement of the ulnar nerve have been provided in the reconstructive surgery of the late war. As a result of this collateral experience in peripheral nerve surgery, the technique of anterior transposition has been standardized, and the one essential feature is the embedding of the nerve deeply in an intramuscular plane. This operation has gradually established itself as the ideal procedure for the treatment of delayed ulnar neuritis, and has been advocated amongst others by Dean Lewis and Miller, Adson, and Buzzard. It satisfies the primary requirements, viz., to shorten the course of the nerve, and to place it in a bed where both friction and tension are eliminated. In my own hands the operation has proved an unqualified success, and in my judgement is to be regarded as the routine procedure for all cases of late ulnar-nerve palsy.

#### SUMMARY.

1. Late involvement of the ulnar nerve (delayed ulnar palsy) is a characteristic remote sequela of certain fractures of the *external condyle* sustained in early childhood.

2. *The clinical picture embraces three phases:* (a) The fracture in early life; (b) The latent period—this is rarely less than ten years; (c) The development of neuritis.

3. The neuritis is a friction or tension lesion. It is pre-determined by the existence of a *gross cubitus valgus deformity*. The nerve-trunk is compelled to take a longer course, and becomes stretched like a bowstring in the shallow post-condylar groove.

4. The onset of symptoms is often associated with a period of strenuous use of the limb in which repeated flexion movements of the elbow are carried out.

5. Operative treatment is indicated in all cases. It is necessary to relieve the nerve from friction, and to shorten its course. Two procedures are worthy of adoption, viz.: (a) Anterior transposition of the nerve. This is an operation of great precision, easy to perform, and has proved completely effective. (b) Supracondylar osteotomy. This is designed to correct the cubitus valgus deformity and so indirectly shorten the course of the nerve. It presents more difficulties than simple anterior transposition.

### 3. RECURRING DISLOCATION OF THE ULNAR NERVE.

Minor degrees of hypermobility of the ulnar nerve are often discovered in the course of routine examinations of the elbow region, but few individuals develop a true complete dislocation of the nerve from its groove. Of the predisposing causes of hypermobility or luxation little is known. Variations in the depth of the post-condylar groove, congenital or acquired laxity of the nerve sheath or arcuate ligament, and, more especially, an exaggerated 'carrying angle', have all been cited as possible explanations (Stopford<sup>12</sup>). It is true that in a number of the recorded cases an increase in the normal degree of cubitus valgus appears as a definite clinical feature (Schmidt<sup>13</sup>), but it is difficult to explain why this factor *per se* should favour the occurrence of luxation. Although a good many cases have been submitted to operation, the information derived from such a source on this question of etiology has been somewhat indefinite. Thus, in three out of six operations for this disability recorded below (*Series IV*), a genuine laxity of the nerve 'mesentery' could be demonstrated, but the exact significance of this anomaly seems obscure. It is evident that an unusually loose anchorage of this type is present throughout life in a certain proportion of individuals. The first dislocation may then be induced by a single severe trauma. In most cases, however, the luxation develops insidiously and may be unsuspected for a considerable period. The onset of neuritis in a nerve which undergoes habitual luxation may be long delayed, there being a silent period as in the late involvement of this nerve after fracture. The neuritis, when it develops, is a typical friction lesion, with the ultimate formation of a nerve spindle (fusiform neuroma).

#### SYMPTOMATOLOGY.

It is unnecessary to enter into an extended description of the symptoms of the 'friction' neuritis induced by a slipping ulnar nerve, as the clinical picture is a faithful parallel of the neuritis of the late involvement. Here again, either sensory or motor symptoms may predominate, and the nerve-trunk becomes thickened and tender as the fusiform neuroma slowly appears.

#### RECORDS OF CASES.

##### SERIES IV. CASES 19-24.

*Case 19.*—J. L., male, age 35 (1923).

**HISTORY.**—Sustained a gunshot wound of the right forearm in September, 1918. Numbness along the inner border of the hand was complained of soon after the injury. A partial lesion of the ulnar nerve was known to be present some years later, but owing to the absence of motor symptoms there was little or no disability. Four years from the date of the wound, signs of an active ulnar neuritis appeared for no apparent reason; the chief symptom was pain and tingling referred to the sensory area of distribution. The symptoms increased steadily in severity.

**CONDITION ON EXAMINATION.**—There was an inconspicuous wound scar on the mesial aspect of the upper third of the forearm. Full ulnar anæsthesia and analgesia were present, but no signs of paresis or wasting of the intrinsic muscles. There was no tenderness of the nerve-trunk in the region of the wound scar. Examination of

the elbow showed that the nerve was hypermobile and slipped forwards in front of the internal condyle with each movement of flexion. The nerve-trunk in the groove was tender and slightly swollen. There was no exaggeration of the carrying angle.

OPERATIVE TREATMENT was decided upon, and the nerve was exposed (a) in the forearm in the region of the old gunshot scar, and (b) at the elbow. In the former situation the nerve was lightly adherent, but showed little or no signs of alteration in calibre. It was accordingly left *in situ*, as its surroundings were considered to be suitable. At the elbow the post-condylar groove appeared abnormally shallow; the nerve-trunk was slightly swollen but showed no induration. It was displaced forwards in front of the elbow, and buried according to the usual technique.

*Result.*—Disappearance of the irritative signs within a few months. The anaesthesia and analgesia have remained unchanged.

*Case 20.*—R. W., male, age 30 (1923).

HISTORY.—Sustained a gunshot wound of the right forearm in 1917 with a compound fracture of the ulna. Healing was slow, and a good deal of stiffness of the fingers remained. There was no evidence of any ulnar-nerve involvement.

*Six years later*, whilst undergoing a course of training in carpentry, the patient began to feel pain on the inner side of the elbow. This pain was best marked during strenuous efforts at planing and similar manoeuvres.

CONDITION ON EXAMINATION.—This revealed a luxable ulnar nerve at the elbow. The nerve-trunk was tender and slightly thickened. There was no increase in the carrying angle.

Conservative treatment was advised and tried for a time; with modified working conditions the pain and paraesthesia diminished. Attempts to resume active work brought on the symptoms once more. Operative treatment was therefore recommended and accepted.

OPERATION.—Anterior displacement of the nerve. In the groove the nerve-trunk was found to be congested, slightly swollen, and just a little indurated. The groove appeared to be unusually shallow, but no other anomaly was recorded.

*Result.*—Complete relief of the irritative symptoms followed the operation within a few weeks, and there has been no return.

*Case 21.*—C. T., female, age 27 (1923).

HISTORY.—Whilst working in a mill she sustained a contusion of the left elbow. One month later pain was still present and the patient had become aware of the slipping of some structure on the inner side of the elbow during each movement of flexion. Each slip produced local pain and also pain referred to the little finger.

CONDITION ON EXAMINATION.—A slipping ulnar nerve was found; the post-condylar groove was wide and shallow. The nerve-trunk showed neither thickening nor tenderness, and there were no signs of interference with conduction. In view of the patient's occupation, operation was advised.

OPERATION.—Anterior displacement of the nerve. The fascial covering of the nerve in the groove was abnormally lax and resembled a bursal sac.

*Result.*—Complete disappearance of the irritative symptoms within a few weeks. Patient resumed work and there has been no recurrence of symptoms.

*Case 22.*—W. J. L., male, age 40 (1924).

HISTORY.—Patient complained that for the past six months he had felt something slip on the inner side of the elbow. This occurred during work, and was accompanied each time by a jarring sensation and pain referred along the inner border of the little finger.

CONDITION ON EXAMINATION.—An inspection of the elbow revealed a lax ulnar nerve which slipped forward on to the internal condyle during each movement of flexion. The nerve was tender and somewhat thickened. The faintest trace of

interference with conduction was demonstrated, i.e., hypo-aesthesia in the little and ring fingers, and slight flattening of the hypothenar eminence. There was no increase in the carrying angle. Operative treatment was advised and carried out.

**OPERATION.**—Anterior displacement of the nerve. The nerve-trunk showed no definite enlargement or induration; the very thin fascial sheath seemed unduly lax.

**Result.**—Complete relief of the irritative symptoms.

*Case 23.*—J. L. D., male, age 32 (1924).

**HISTORY.**—Sustained a gunshot wound of the left forearm in 1918, with an incomplete involvement of the ulnar nerve which was not treated by operation. Four years later the nerve at the elbow became painful and tender, and it was found to be hypermobile. Conservative treatment was tried for nearly a year, but the irritative symptoms persisted. Operation was therefore suggested.

**OPERATION.**—Anterior displacement of the nerve. No striking anatomical anomalies were noted.

**Result.**—Very marked improvement, but incomplete relief of the irritative symptoms. It was considered that the residual signs were caused by the old gunshot lesion in the forearm.

*Case 24.*—W. A. C., male, age 33 (1924).

**HISTORY.**—Sustained a contusion of the left forearm which was followed by persistent pain. Pain was referred to the little finger, and the patient was conscious of something slipping.

**CONDITION ON EXAMINATION.**—A hypermobile nerve was discovered which dislocated with every movement of flexion. The nerve was tender, but there were no signs of interference with conduction. It was doubtful whether the symptoms were associated entirely with the slipping nerve: but as the patient had become increasingly aware of the recurrent luxation, it was thought wise to operate. There was no increase in the carrying angle.

**OPERATION.**—Anterior displacement of the nerve. The nerve appeared to be slightly flattened, and the hypermobility was due to an unduly lax covering sheath; this was almost mesenteric in its attachment.

**Result.**—Complete abolition of the irritative symptoms in the elbow region.

## TREATMENT.

**1. Recurring Dislocation without Neuritis.**—In cases where the luxation has been recently discovered and before the onset of signs of definite neuritis, it is advisable to give a trial to conservative measures unless there are strong economic reasons for urging early operation. If it is possible to enjoin a limited use of the elbow with the avoidance of repeated forcible movements of flexion, the stage of neuritis may be postponed almost indefinitely. Operative treatment should be considered, however, in individuals who are engaged in manual occupations, or in others who are unable to submit to the necessary restrictions demanded by conservative treatment.

**2. Recurring Dislocation with Neuritis.**—With the appearance of the first symptoms of neuritis, conservative treatment has little to offer; in such cases operation should not be delayed. Various elaborate procedures have been employed in the past: but there can be little doubt that there is one type of operation only which is applicable for this lesion, viz., *anterior transposition* of the nerve-trunk. The objections to the older or alternative operations have already been fully stressed. As in other forms of traumatic neuritis of this nerve in the post-condylar groove, the operation of anterior displacement is followed by results which are exceedingly gratifying.

## THE OPERATION OF ANTERIOR TRANSPOSITION OF THE ULNAR.

A full description of this operation was given by Stiles<sup>14</sup> some years ago, and more recently by the writer in Carson's *Modern Operative Surgery*.<sup>15</sup> It seems worth while to consider here quite briefly the main technical features of an operation which has proved to be so effective in the treatment of several types of traumatic ulnar neuritis, and also has a much wider application. I have now employed this procedure on some 90 occasions.

As already mentioned, the operation came into prominence during the hey-day of the peripheral nerve surgery of the war. The indications for its employment may be summarized as follows:—

1. As an aid to *end-to-end* suture in gross lesions of the nerve with extensive loss of continuity. Thus a series may be quoted of 115 ulnar-nerve sutures for gunshot lesions performed by the writer, in 55 of which it was necessary to displace the nerve in front of the elbow.<sup>16</sup>

2. As a form of *neurolysis* for all incomplete lesions of the nerve in the post-condylar groove: (a) Gunshot wounds and other severe traumata; (b) The various types of ulnar neuritis described in this paper. It may be stated here, as a fundamental principle, that when it is necessary to explore at operation an incomplete lesion of the ulnar nerve in the post-condylar groove, the nerve-trunk should never be returned to its original bed. In all such cases this bed is now unsuitable, and the nerve should be displaced forwards and buried deeply where it is no longer exposed to abnormal tension or friction. This is the ideal neurolysis.

The operation of anterior displacement comprises the following steps: (1) Generous freeing of the nerve-trunk in the lower third of the upper arm, in the groove, and in the upper third of the forearm. In the latter situation access is gained by splitting the line of fusion of the two heads of origin of the flexor carpi ulnaris. (2) The proximal branches of the nerve must be carefully freed to allow the nerve-trunk to be fully mobilized. It is necessary to sacrifice the fine branch to the elbow-joint, but great care must be taken to conserve the branches to the flexor carpi ulnaris and flexor profundus digitorum which arise in the lower part of the groove. These twigs are stripped up in a proximal direction from within the sheath of the parent trunk, and also stripped distally to increase their extramuscular course. In this way kinking is avoided, and the ulnar trunk can be easily displaced well in front of the condyle. (3) The new nerve-bed must be situated deeply in an intramuscular plane. It is constructed either (a) by division of the superficial fibres of the forearm flexors taking origin from the internal condyle, or (b) by the detachment and turning down from the condyle itself a tongue-shaped muscular flap (Bristow). (4) Where the nerve-trunk passes forwards in the upper arm across the line of the intermuscular septum, this structure must be excised over a sufficient area. Unless this is done, the nerve is likely to be bow-stringed over a tense fascial bridge.

## SUMMARY.

1. There are certain special types of ulnar-nerve injury which are primarily determined by an alteration in the normal relation between the



nerve-trunk and its bed in the postcondylar groove. Under such conditions the nerve becomes exposed to the cumulative trauma of repeated overstretching or friction.

2. The nerve lesions produced in this way are ordinarily of the incomplete type, and may be described under the broad title of traumatic neuritis.

3. Three clinical groups may be distinguished: (a) Ulnar-nerve injuries associated with recent fractures of the lower end of the humerus. In these, spontaneous recovery is the rule; operation is called for in rare cases of severe and persistent neuritis. (b) Late ulnar-nerve involvement after fractures. (c) Recurring dislocation of the ulnar nerve. In the two latter groups operative treatment is indicated as a routine.

4. There is one simple and effective operation which is universally applicable in all cases of traumatic neuritis of the ulnar nerve in the postcondylar groove, viz., *anterior displacement* of the nerve-trunk.

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## OPERATIVE REPAIR OF CRUCIATE LIGAMENTS IN SEVERE TRAUMA OF KNEE.

By ALEXANDER H. EDWARDS, GLASGOW.

### OPERATION.

AN incision is made commencing over the centre of the lateral condyle

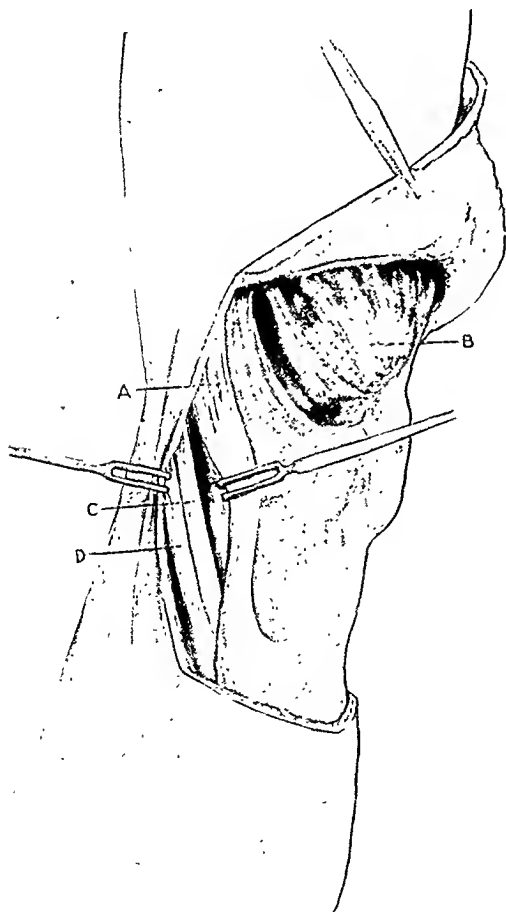


FIG. 274.—Showing skin and subcutaneous tissues reflected to expose tendons. A, Sartorius; B, Vastus internus; C, Gracilis; D, Semitendinosus.

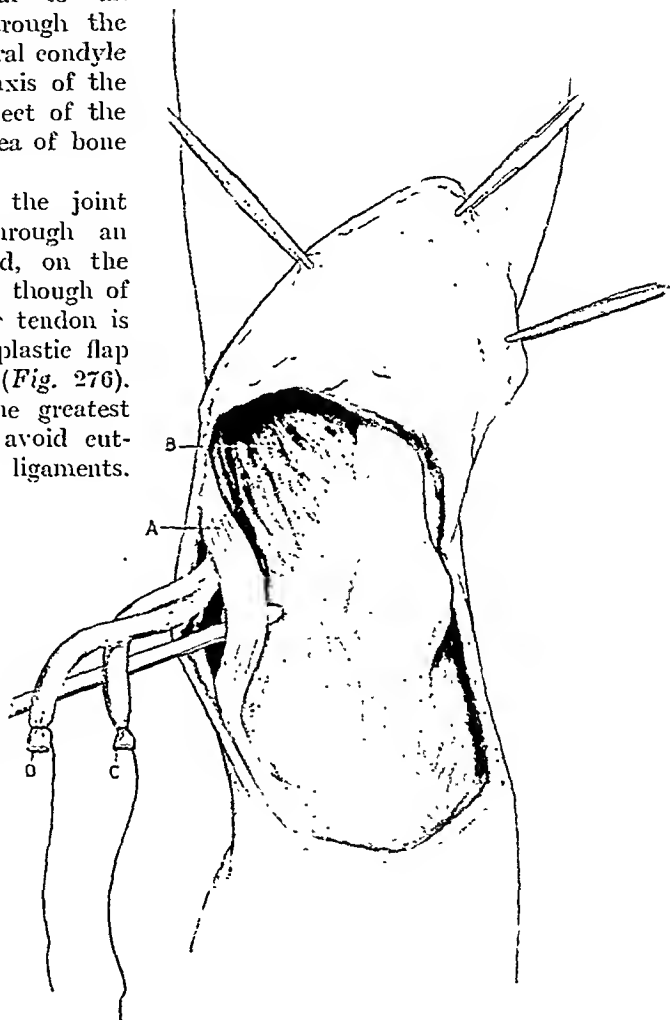
of the femur. It passes at first distally and parallel to the long axis of the limb, but soon curves dorsally, keeping about one inch lateral to the anterior tubercle of the tibia and the same distance beyond this landmark. From the latter point it curves in a proximal direction behind the medial condyle of the femur. At the level of the centre of this condyle it passes proximally parallel to the axis of the limb, and ends at a point or level 3 or 4 in. from the proximal margin of the patella. The incision is roughly of horseshoe shape. The skin and subcutaneous tissues are to be reflected to the fullest extent of the incision, after which the sartorius muscle is clearly defined, and drawn aside to allow free access to the gracilis and semitendinosus tendons (*Fig. 274*). These should be dissected with their sheaths intact. After they have been exposed in their entire length, they are divided close to their insertions (*Fig. 275*). The ends are then grasped in forceps, covered with protective gauze, and laid aside till a later stage.

The next step is to divide the deep fascia and attachments of the vastus medialis to the proximal (upper) surface of the medial

condyle. The bone at this point is to be cleared with a detacher, so as to allow drilling of a passage from the newly cleared area through to the centre of the joint cavity. The area covered by the point of the drill is immediately ventral to the adductor magnus tendon. Next the deep fascia close to the medial tuberosity of the tibia is exposed, and the bone here laid bare, slightly dorsal to an imaginary line passing through the centre of the medial femoral condyle and parallel to the long axis of the limb. On the lateral aspect of the tibia the corresponding area of bone is exposed.

A free exposure of the joint cavity is now made through an incision, horseshoe shaped, on the lines of the first incision, though of less extent. The patellar tendon is thrown up with an osteoplastic flap from the tibial tubercle (*Fig. 276*). In exposing the joint, the greatest care should be taken to avoid cutting either of the lateral ligaments. These should be examined for signs of laceration, and if possible repaired after the cruciate ligaments have been dealt with.

A drill,  $\frac{1}{4}$  to  $\frac{3}{8}$  in. diameter, 4 to 5 in. long, for the tibia, and  $\frac{3}{8}$  to  $\frac{1}{2}$  in. diameter for the femur, should now be requisitioned to make the new bone canals necessary in the femur and tibia. The tibia should be drilled on each side, the point of the drill being applied to the areas already prepared, and the lines converging in a proximal direction to meet



*FIG. 275.*—Showing tendons divided. A, Sartorius; B, Vastus internus; C, Gracilis; D, Semitendinosus.

in a common opening at the tibial spines (*Fig. 277*). The drilling will probably be best done by hand, as the drill is thus more easily controlled, and with ordinary care the proper working angles can be maintained. It will be necessary to have the knee slightly flexed to complete this part of

the operation, but for the drilling of the medial condyle the flexion must be full. The new canal begins at the part close to the adductor magnus tendon already mentioned. As a guide to the angle, a long thick probe may be passed through the opening in the lateral tuberosity of the tibia, and the line to follow is that towards the centre of the intercondylar space, or slightly dorsal to this. All bone débris should now be cleared from the canals made and from the joint cavity; the tendons are then uncovered,

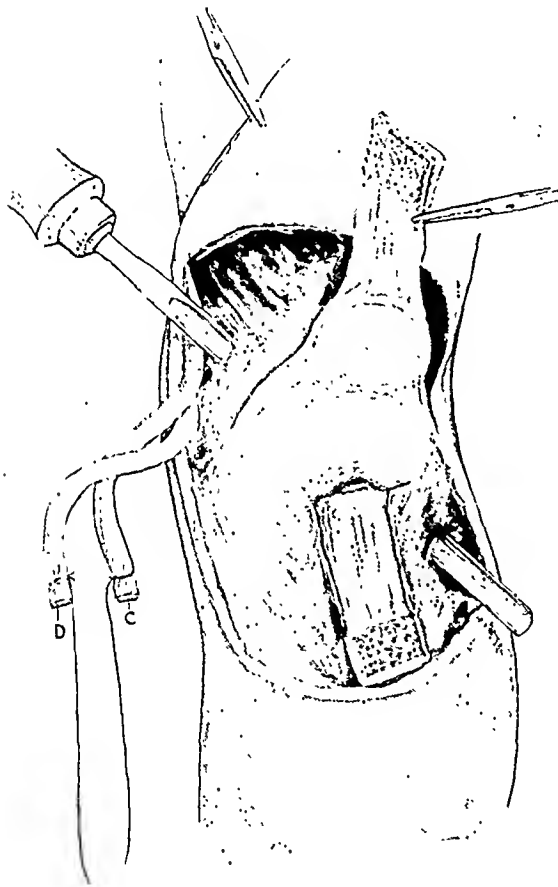


FIG. 276.—Exposure of knee-joint by raising patellar tendon with an osteoplastic flap. C, Gracilis; D, Semitendinosus.

and long silk sutures fixed securely through their distal ends. Long forceps are pushed through the femoral opening from the joint side, and by means of them and the silk ligatures the tendons are pulled into the joint. The tendons are now crossed, or twisted (*Fig. 278*), the semitendinosus being drawn through the lateral tuberosity of the tibia, and the gracilis passed through the other artificial canal (*Fig. 279*). They are now pulled as tight as possible with the knee fully extended, after which, the tension being still maintained, they are fixed down securely with silk to the deep fascia of their respective areas. The osteoplastic flap is returned to its place and nailed down, the joint capsule being carefully sutured thereafter (*Fig. 280*). After closure of the superficial wound, the knee should be fixed on a splint in slight flexion till healing is complete, and as soon as possible thereafter a walking plaster applied to include the foot, knee, and pelvis.

The operation just described was examined on the cadaver by Professors Bryce (Glasgow) and Waterston (St. Andrews) in their respective Anatomy Departments. Both of these eminent authorities expressed approval of it as an anatomical proposition, whilst Mr. J. Hogarth Pringle, F.R.C.S., lately surgeon to the Royal Infirmary, Glasgow, expressed equal approval from the surgical standpoint.

## REMARKS.

The above operation was carried out experimentally in the Anatomy Department of the University of St. Andrews in 1918, with the very kind permission of Professor Waterston. No opportunity for its practical application has occurred since that year. It is published in the hope that in some cases it may be found useful. In the course of some years of work in examining pensioners, the writer has seen a number of cases of flail knee. Throughout the United Kingdom, and in all countries involved in the Great War, there must be a large total number of patients suffering from this disabling condition. The operation of ankylosis of the knee has been frequently done, and this classical procedure may have been for the surgeon the simplest way out of the difficulty; but it may not have been the ideal solution, for even a perfect ankylosis still precludes the possibility of the patient following certain common occupations, thereby diminishing the wage-earning average.

The object of this paper is not to discuss primary treatment, operative or conservative, but to attempt to offer a scheme of constructive surgery which may be of service in cases of flail knee resulting from destruction of the cruciate ligaments. In such cases, where conservative treatment has

failed, it is hoped that surgical interference on these lines may be considered. Jones and Lovett, in their text-book of orthopaedic surgery, state that they have examined several cases of "old flail knees with marked functional defect", and after reconstructive operation they had not seen a perfect result, though several had been much improved.

Various reconstructive methods have been devised. In one, the iliotibial band is transplanted, and an attempt made to convert it into an intra-articular ligament (Aylwin Smith). It is thought that a serious objection

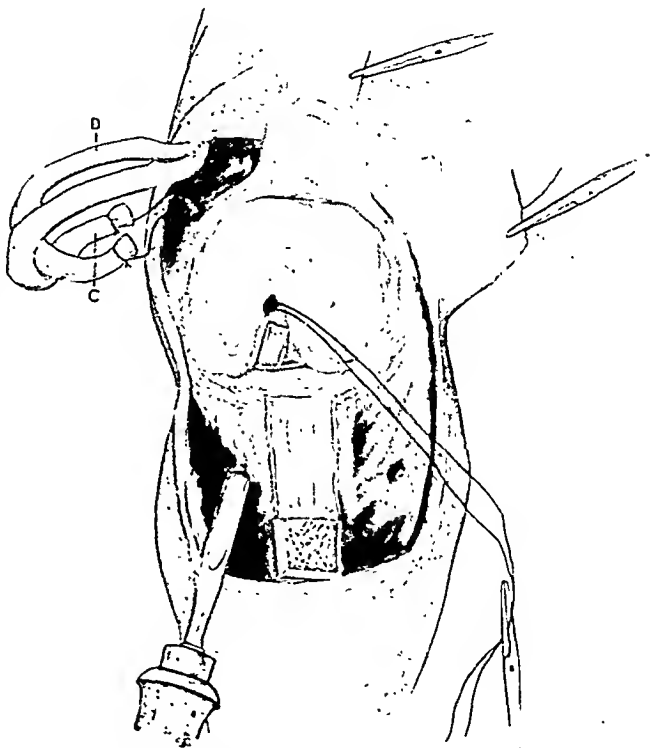


FIG. 277.—Showing the holes drilled in the bones through which the tendons are passed. C, Gracilis; D, Semitendinosus.

At the Leeds Infirmary up to 1912 the presence of these glands in abdomens opened for operations of various kinds was practically unrecorded. As a result of a request by the author, the numbers noted began to increase year by year, until in 1924 77 were noted.

Out of 58,731 in-patient cases of all kinds operated upon at Leeds General Infirmary between 1912 and 1924 (inclusive), only 435 were stated to have tuberculous abdominal glands, made up as follows :—

	MALES	FEMALES	TOTAL
Cases operated on ..	35,167	23,564	58,731
Number with tuberculous mesenteric glands ..	228	207	435

These are made up as follows :—

AGES	MALES	FEMALES	TOTAL
Up to and including 10 years ..	46	38	84
Over 10 but not over 20 ..	80	67	147
"    20    "    "    30 ..	49	47	96
"    30    "    "    40 ..	26	32	58
"    40 years of age ..	27	18	45
No age given ..	—	5	5
	228	207	435

As showing the effect of a personal interest, more than one-third of these cases were under the care of the author of this paper.

The difficulty in arriving at a true estimate is again shown in an extraordinary diversity in the figures in different areas. As is well known, surgical tuberculosis is found more often in certain areas in Scotland than elsewhere. It is not surprising to find that the disease is rife in a very limited area and apparently absent in areas contiguous to this. In the author's experience a series of cases has twice been traced to one 'milk round'; in both infected cows were found to be the source of the trouble.

It is remarkable to find that the percentage of tuberculous abdominal gland cases is higher in private than it is in infirmary practice. The author has himself operated upon over one hundred cases in private practice, and about an equal number in the very much bigger infirmary practice.

Corner believes that tuberculous mesenteric glands are to be found in practically every child in which an abdominal operation is necessary.

Risley believes the disease to be extremely common in infancy and childhood but by no means confined to this period, being nearly as common in young adults. In fact, the highest percentage favours the years from 16 to 18.

Eisenhardt, in 1000 post-mortems of adult phthisis cases, found that 563 had intestinal tuberculosis; Ronseff in 800 found 46 per cent; and Harnan, in 832, 68 per cent.

Struthers says in the years 1919–20 he met with 22 cases of mesenteric adenitis, whilst he dealt with 187 cases of appendicitis; most of the cases were young children.

Shiota, reporting 24 cases from Japan, makes the interesting observation that *none* of them had drunk milk.

Franke in 1914, at the forty-third Surgical Congress in Berlin, reported 90 cases in which operation measures were taken.

## TUBERCULOSIS OF ILEOCÆCAL GLANDS 441

Maclaren in 1916 reported 100 of these cases during four or five years, some of them sent for the purpose of having their appendices out.

Frankel, of the North Chicago Hospital, writing in 1917, says: "All statistics show the rarity of intestinal tuberculosis in the first year of life, the frequency of the disease gradually increasing until the fourth or fifth years, and then diminishing again. This term coincides with the average time of weaning and change from mother's milk to the diet through which contaminated food has an opportunity to invade the intestinal canal".

It is interesting to find that according to Scott, of Washington, only 5 per cent of all appendices removed are tuberculous; whilst according to Lockwood there are 2 per cent, and according to Tetz the figure is 3.11 per cent.

Dr. Dingwall Fordyce, writing of the Royal Hospital for Sick Children, Edinburgh (1909), says the disease is extremely common—in the Edinburgh district at least.

Goodhart and Still say that although 59 per cent of all tuberculous post-mortems showed tuberculosis of mesenteric glands, only 46 cases were diagnosed clinically in 6000 to 7000 patients—half of them being very doubtful.

Carson gives the age incidence as follows:—

CASES				CASES			
Between 1 and 5	..	8		Between 15 and 20	..	2	
" 5 " 10	..	14		" 20 " 25	..	4	
" 10 " 15	..	15		" 25 " 30	..	5	
				Over 30	..	2	

Of the 50 cases, 29 were males and 21 females.

Corner (1905) says mesenteric glands are the most frequent source of tuberculous infection of the body. In 1912 he writes that in children with abdominal signs there are cent per cent of tuberculous abdominal glands. He quotes Professor E. Sims Woodhead as finding 78.7 per cent in autopsies on tubercular children.

Dr. Sabourin, of Durtol, in 1913, makes the very interesting observation in respect of sanatorium patients that in each batch of 50 boarders 20 to 25 suffer in respect of their 'appendix'—i.e., symptoms in the right iliac fossa.

Opie, of the Washington University (1920-1921), says few instances of healed tuberculosis of the mesenteric lymph nodes are found among children or adults examined in the city of St. Louis. In a series of 93 autopsies made in 1917 upon children, and of 50 autopsies made on adults, no instance of healed mesenteric tuberculosis was found. Nevertheless it is probable that primary tuberculosis of the gastro-intestinal tract occurs in a small proportion of children in St. Louis, as elsewhere. An impressive contrast has been furnished by autopsies performed on British soldiers at Base Hospital 21, stationed at Rouen, France, for they have demonstrated the surprising frequency of healed calcified, occasionally still partially caseous, or even frankly caseous, lesions of the mesenteric lymph nodes. Healed focal and lymphatic tuberculosis of the lungs similar to that seen in St. Louis was common, and often the lesions were very extensive; but instances in which little of any old pulmonary tuberculosis was demonstrable appeared to be

much more common among the British than in St. Louis, where the former observations had been made. It is not improbable that the lower incidence of the lesions of the lungs bears some relation to the greater frequency of localized infection by way of the intestinal tract.

Caseous or calcified mesenteric nodules were found in 18 instances among 66 young male adults, whose ages, with few exceptions, varied from twenty to thirty years. Among 15 autopsies, of which notes were lost, the proportion of mesenteric tuberculosis was approximately the same. In a number of instances X-ray plates were made from both the mesentery and the lungs, and they serve to show with considerable accuracy the size and position of the calcified lesions which were present.

### SYMPTOMATOLOGY.

It is convenient to consider at this point whether a non-tuberculous adenitis of the ileocaecal glands is a condition likely to occur and to give rise to symptoms.

There is no doubt that in animals, especially cats, simple ileocaecal adenitis is exceedingly common; in man a much greater immunity from adenitis is likely, but it is fair to suggest that the condition may occur.

In the *BRITISH JOURNAL OF SURGERY* (July, 1923) the author writes of ileocaecal adenitis and its possible bearing on the causation of duodenal and gastric ulcer. Is it not likely that some of the pain so commonly complained of in the right iliac fossa may be due to a simple inflammatory condition of these glands, infected from the contiguous bowel?

The symptomatology of tuberculous ileocaecal glands divides itself into two definite groups: (1) *In children*; (2) *In adults*.

The first group comprises two sections (*a*) *acute*, (*b*) *subacute*, which depend upon the degree of acuity of the adenitis. The second group is to be considered as the chronic stage with subacute exacerbations. It will be clear that no definite line can be drawn between these varieties, because the acute is sometimes, though very rarely, met with in adults, while the chronic is sometimes seen in children.

Speaking broadly, *Group 1* consists of cases with an acute or subacute adenitis, whilst *Group 2* includes only cases in which, natural repair or resistance having been achieved, the resulting products, i.e., caseation and calcification, are responsible for the symptomatology and physical signs. It follows, therefore, that *Group 1* is not commonly capable of a diagnosis by X rays, whereas *Group 2* usually is.

*Group 1.—Children from 2 to 10 Years.*—It is a fallacy to suppose the child bears the usual stigmata of tuberculosis. In cases where the tuberculosis diathesis is typical, i.e., in children of poor parents and living in unhygienic conditions, it is much more common to find the disease involving not only the glands but the mesentery, the omentum, and the last part of the ileum. The typical case of tuberculosis of ileocaecal glands is one where the child is fat, phlegmatic, and pallid, usually of good parentage, and living in perfectly healthy surroundings. As a rule there has been no history suggesting tuberculosis anywhere; occasionally, however, one hears of cervical glands which have given rise to anxiety.



*a. Acute*—In children from 2 to 6 years.—The onset can only be compared with that of an acute appendicitis. It is sudden and violent; the child draws up its legs, the abdomen is rigid, and tenderness, although present all over it, is chiefly recognized in the right iliac fossa. The pulse rises rapidly to 120, the temperature to 100° or even to 103°; respirations are increased in number, but more shallow in excursion. Vomiting ensues and the tongue becomes furred. Is it possible for anyone under these circumstances to take the risk of putting aside the possibility of acute appendicitis?

It has been suggested that an acutely inflamed gland causes a rise of temperature, whereas acute appendicitis does not. How many men in general practice will agree with this? It is known that almost every acute painful condition in a child gives rise to an elevation of temperature. A temperature above 100° would indicate the condition to be tuberculous adenitis rather than appendicitis.

Painful micturition, especially pain at the end of the act, is certainly to be found in most cases of acute appendicitis; but is it not likely, in order to have this symptom well marked, that the appendix must lie in close contact with the bladder?

Operation should therefore be undertaken for acute appendicitis with a reservation that the condition may be an acute adenitis; sometimes both conditions are present.

If there is present a serious degree of pain with a rigid abdomen, tender chiefly in the right iliac fossa, and if this condition lasts for twelve hours, operation should be undertaken on the assumption that acute appendicitis may be found. It is perfectly well realized that acute attacks of pain in children are common enough. Many are cured by a dose of castor oil; a few are lost; it is for the few, maybe the very, very few, that one pleads.

Assuming that operation is undertaken, what is found when the appendix is not apparently the cause of the trouble? Unless the ileocecal and the mesenteric glands which drain the last six inches of the small intestine are examined, nothing will be found, and the case will be considered one of appendicitis without any obvious change in the organ or one of 'thrombosis of the appendical vessels', or a 'stenosis' will be found in its lumen. There is no doubt in many cases an acute adenitis is present, and is shown by enlargement and redness of this group of glands. Sometimes it is shown by a red areola invading the mesentery along the circumference of one or more.

One may quite well imagine a condition of this kind to result from a simple intestinal toxæmia; but the association of exactly similar changes in the same group of glands with areas of caseation and calcification makes it clear that many at least are tuberculous. It is not suggested that anything more than appendicectomy should be attempted. Probably in a very few cases this is the primary focus; but it is more likely by far that the condition arises as the result of infection in the last few inches of the small intestine, and the starvation and general treatment which follow an abdominal operation cure the acute condition.

*b. Subacute*—Usually in children from 6 to 10 years. Similar in type to those in (a). In this condition we are dealing probably with the repair of a gland, and not, as in (a), with the onset of the disease. The symptoms are

somewhat similar but less marked: repeated attacks of sudden abdominal pain, suggestive of intestinal colic (in fact they may be attacks of colic).

With the onset of pain the child cries out, holds its belly with both hands, draws up its legs, and in ten minutes is perfectly fit and well. Occasionally there is vomiting; more rarely there is a passing rise of temperature to  $100^{\circ}$ . The child, though appearing in pain during the attack, is perfectly well immediately after and before. Deep palpation may reveal a tender spot in the region of the appendix. There may be two or three attacks in a day, or there may be intervals of months between each attack.

Again the question of appendicitis arises! Can anyone in our present state of knowledge be certain that this is *not* appendicitis? It is true that in this group of cases one has more time to think, and operation is not so clearly indicated as in (a); but unless the child is under close observation the assumption of tuberculous adenitis is a risky one.

As a rule these cases are diagnosed as 'appendicular colic' or possibly 'subacute appendicitis', and operation is usually undertaken deliberately in an interval. A perfectly normal appendix is found and removed; the absence of any inflammation is accounted for by the fact that only an 'appendicular colic' is being dealt with, or a 'concretion in the lumen' is held to be sufficient cause. Unless examination is made of the glands in the ileocaecal angle and in the mesentery of the last six or eight inches of the small intestine, the real cause of the pain will remain undiscovered, and recurrence of the attacks will be certain. Removal of the affected gland will ensure a cure of the condition; but even if it is not removable, or if the condition is widespread and therefore probably inoperable, one will be in a position to explain to the parents that recurrence of pain may occur, and one will also be able to assure them that, although the child may have pain, the origin of the pain is known, and if the appendix is removed there is nothing left which could give rise to a fatal issue.

It is, of course, perfectly recognized that, assuming the condition is left alone, a natural cure will eventually take place.

Diagnosis in this class may quite well be made by an X-ray examination; and assuming this to be the position, there would be some ground for holding one's hand as regards operation, though there would be considerable risk to be taken in doing so, and considerable anxiety to everybody concerned.

Between the ages of 10 and 15 years there appears to be a period when the disease is not commonly evident.

**Group 2.—Adults from 15 to 45 Years.**—This type represents the common case of *chronic* aching pain in the right iliac fossa; it is slightly more common in women than in men. The patients as a rule are thin and anæmic, usually constipated and of a 'nervous temperament'; sometimes they are apparently perfectly well, fat and ruddy, and certainly not nervous.

The complaint of pain is over a period of years, with long intervals of freedom. The site of the pain is about MacBurney's point, sometimes above it, but rarely below; it is a dull ache, a drag, or occasionally a stab. When the gland is pressing deeply into the iliac fossa there is sometimes pain

suggestive of renal disease, and sometimes pain increased by full extension of the leg at the hip, either on walking or playing active games, such as tennis. Relief is sometimes achieved by lying down, though in a bad attack this may be by no means the case. There are no constitutional disturbances, and there is no rise of temperature.

It would appear that the cause of the pain is pure irritation, a peritonitis at the surface of one or more glands, due to the presence of caseous material, or to actual impinging of calcified spicules on to the peritoneal covering. In the latter case, the white spikes of lime salts can sometimes be seen lying naked, pushing their points through a surrounding pink or red areola; the spikes are often 'flaked' with red shreds of organized lymph. Tenderness is *always* present, absolutely localized to the area of the pain.

Many of these cases are called 'neurotics'. They are a misery to themselves and to their doctors. Operations are performed with a diagnosis of appendicitis, and a *tiny opening is made through which the tip of the cæcum is produced, the appendix is removed, and a beautiful scar is left.* Any subsequent pain is said to be due to adhesions, and sometimes further operation is performed for the division of these. Massage is a favourite device; naturally it is a very painful business, but the patient (being neurotic!) is advised to persist with it. Relief can only come by a very prolonged process of natural cure or by the removal of the offending source of irritation by operation. Diagnosis can be made in nearly all these cases by X-ray examination.

It will be useful here to recapitulate what has been said by numerous authors on the symptoms and diagnosis of the condition, with special reference to its simulation of appendicitis.

RISLEY<sup>1</sup> says that the clinical picture is that of an acute right lower quadrant condition simulating appendicitis. There are two clinical types: (a) Distinctly palpable masses of glands with vague or no abdominal symptoms; and (b) Cases with alarming abdominal symptoms and signs developing suddenly when there are no palpable glands demonstrable. Diagnosis is often difficult or impossible.

*Symptoms:* (a) Trivial, fleeting, and not severe right-sided or general abdominal pain with or without disturbance of digestion and bowel regularity, the general health being good, or patient of a pale, rather sickly type. (b) More or less constant fairly localized abdominal pain, with a soreness referable to the cæcum. (c) Sudden (or gradual) onset of generalized, then localized, pain in the right lower quadrant. Temperature 99°, 100°, or 101°; nausea, vomiting, constipation or diarrhœa, with local tenderness, spasm, and resistance; white count 12,000 or 15,000; very sick appearance. There may be distention.

The process may be fulminating, and an immediate operation may save patient from general tuberculous peritonitis.

CAUSON<sup>2</sup> says the condition is probably much more common than is supposed, most cases being labelled 'indigestion'. He describes under-developed patients, not gaining weight and strength, never been strong; thin, listless, appetite poor, tongue furred; only a few cases of rise in temperature (99° to 100°); pulse only quick in pain. The main sign is pain—*absolutely typical* in character—"Sudden centralized abdominal pain severe enough to make the child cry, lasting fifteen minutes or less, relieved by pressure and hot applications, recurring perhaps two or three times a day, and stopping as suddenly as it began, so that in the intervals the patient is quite free". He thinks it is a true colic. He believes the gland stimulates the neighbouring vagus nerves proceeding to Auerbach's plexus, and causes a tonic contraction of the circular muscle-fibres of the small intestine, and

that the pain is due to this cause. In three cases he saw an intussusception. Can this cause the pain?

CARSON<sup>2</sup> also says it is rare to find any lump; there is no free fluid, no rigidity, no distention. In a few cases he has had an X-ray, but only once has he seen a vague shadow of a caseating mass.

CORNER<sup>3</sup> says that the children suffer from ill health, and abdominal pain referred to the umbilical region. These pains come on at night, and sometimes after food. There is loss of appetite, anorexia, often accompanied by some disturbance of the action of the bowels, more often inactivity and constipation than looseness of the bowels.

STRUTHERS<sup>4</sup> says:—

“Within recent years a number of cases have come under observation in which patients apparently suffering from appendicitis have turned out on operation to be suffering from lymphadenitis; that is to say, the appendix has been found perfectly healthy, and the only lesion detected has been enlargement of the lymph glands in the mesentery, with signs of peritoneal irritation over them. In most of the cases, but not in all, the enlargement has obviously been due mainly, if not altogether, to tuberculosis; but whether the acute symptoms simulating appendicitis have been due to the tuberculosis alone, to tuberculosis with super-added transient infection of another kind, or in some cases to non-tuberculous lymphadenitis alone, it is difficult and, in the meantime, almost impossible to say. . . . The attacks are relatively mild in character, with moderate fever and constitutional disturbance. The general appearance is not that of severe illness, and in particular the tongue is not heavily furred, nor has the breath the peculiar and almost characteristic odour associated with appendicitis. As a rule the acute symptoms tend to subside in a day or two if the case is watched. During the acute stage the glands are not often or readily felt, owing to the muscular resistance present. Under an anæsthetic, however, they may be more easily palpated, and when several are adherent to each other the resulting lump tends to be higher and nearer the middle line than the swelling caused by the inflamed appendix with omentum or intestine adherent to it. After an acute attack the persistence of a lump and its situation may clear up the diagnosis. It must be admitted, however, that a positive diagnosis as between appendicitis and mesenteric lymphadenitis often cannot be made. Appendicitis is a treacherous disease, and in doubtful cases a ‘wait and see’ policy is apt to be followed by disastrous results, especially in children. One thing may be asserted with confidence, namely, that opening the abdomen in cases of lymphadenitis does not appear to influence the glandular affection unfavourably. The patients do very well, and I have not yet seen a case in which apparent extension of the tuberculous disease has followed operation.”

PARKER<sup>5</sup> says:—

“Unfortunately the diagnosis of this disease is very difficult, and in some cases even impossible, as it apparently follows no definite peculiar symptom-complex. A certain proportion of cases complain of vague abdominal pains pointing to no particular organ, in children possibly coming on at night or after the ingestion of food, accompanied by the loss of appetite and disturbance of bowel function, more often constipation than diarrhoea. . . . It is a peculiarity of this lymphatic gland disease that it occurs either with moderate pains pointing definitely to no abdominal organ, or appears, and with a certain preference, under the aspects of two of our worst and most frequent abdominal diseases. . . . Maylard says: ‘When a palpable tumour is present, the diagnosis is easier than in adults, for where in the latter there are many conditions giving rise to tumours, simple and multiple, within the abdomen, in the former there are but few; indeed, it may be said that hard, movable lumps in the belly of a child, which are not fecal and which remain constant under all conditions, are most certain to be tubercular mesenteric glands’. . . . So it may be truly said that because of the rarity of its appearance, the absolute lack of all indications of tuberculosis in other parts of the body, especially of the intestines, and its peculiar character even in the cases in which a mass is to be plainly felt, a diagnosis can be arrived at only in a conjectural way. In cases of the nature of

those reported, however, from the standpoint of treatment, the more or less accidental pathological diagnosis is not of decisive importance, as laparotomy is indicated anyway, and the condition found can be dealt with as indicated."

MAUCLAIRE<sup>6</sup> says of mesenteric tuberculosis of the lymphatic glands:—

"This form, which is so frequent with children, and constitutes *tabes mesenterica*, has given rise to some tentative operations. This lesion is mostly the result of a chronic evolution. Sometimes, however, it presents acute symptoms which simulate appendicitis, intussusception, etc. Sometimes these inflamed glands suppurate and discharge into the peritoneum or the intestine, which they infect secondarily. In an exceptional case their calcification led to a diagnosis of calculus in the common bile duct."

H. TYRRELL GRAY<sup>7</sup> says of the pain:—

"Inflammation of mesenteric glands, whether acute or chronic, may be associated with colic, which nearly always arises in the small intestine or the ileocolic angle. The primary focus in the intestine may itself be responsible for the pain, as already described; but the glands themselves may also be responsible. For the inhibitory segment of the peristaltic wave normally exerts a physiological degree of tension on the mesentery during its passage, and, in the presence of inflamed glands, drugs on these and causes pain."

SIR JOHN THOMSON-WALKER<sup>8</sup> refers to the frequency of the condition and the rarity of its diagnosis clinically. He states:—

"In 254 necropsies on tuberculous children at the Children's Hospital, Great Ormond Street, 151, or 59 per cent, had tuberculous mesenteric glands, whilst clinically only 46 cases were diagnosed in 6000 or 7000 patients at the Evelina Hospital.

"In a number of the 42 cases other pathological conditions were present in addition to the calcified abdominal glands. Thus in 7 cases there was stone in the kidney or ureter, in 2 there was pyelitis, in 3 urinary tuberculosis, and one patient was pregnant. These conditions all tended to mask the symptoms that might have been due to the glands and thus to confuse the diagnosis. But there were 28 cases in which no other disease beside the calcified abdominal glands could be detected, and the following notes in regard to symptoms are based upon these cases.

"Pain is the chief symptom in calcified abdominal glands, and in my cases it was the prominent feature in 25 of the 28 cases. It was a dull ache in 4, very acute pain amounting to abdominal colic in 14, and moderately acute in 7. In the majority of these pure cases the pain was a severe colic (14 in 25). The duration might vary from a few minutes to several hours. The pain commenced suddenly and usually ceased suddenly. In severity it was comparable to the two chief abdominal colics, namely renal and biliary colic, and was much more severe than that of appendicitis. In distribution it resembled that of moderate renal pain or of renal and ureteral colic in 17 cases, of appendicitis in 4, and the pain resembled biliary colic in one. In 5 the pain area was not defined, and in one case there was no pain. There were certain negative points that helped to distinguish the pain due to calcified glands from that of renal colic. Movement had practically no effect in initiating or in increasing the pain. Vomiting, so common in renal and also in biliary colic, was absent in these cases. There was no retraction of the testicle, and no pain referred to other parts of the body. There was very rarely any disturbance in the action of the bowels, such as might be expected in a case of appendicitis of long standing.

"A tender spot was present in the abdomen in four cases in which the calcified gland was the only disease present. The tender area lay directly over the calcified gland, and in all cases lay within the area of tenderness present in cases of appendicitis, so that this sign tended rather to confuse than to clear the diagnosis. In one case the calcified gland could be felt as a nodule, and rolled beneath the finger at a point in the line of the ureter above the brim of the pelvis.

"The proportion of cases in which the pain resembled that of renal pain, or colic, may have been unduly large from the fact that this class of case was the most likely to come under my care. The explanation of the pain is, I think, to be found in the proximity of the calcified glands to the ureter. The drag or presence of such

a calcareous mass would very easily cause ureteric spasm. The aching pain in the appendix region in 4 cases was due to the position of the calcified gland in this area.

"Blood was present in the urine in microscopic or in naked-eye amount in 6 cases in which no disease except the calcified gland could be detected.

"Any statement in regard to the relation of hæmaturia to calcified abdominal glands must be purely speculative. In the cases in which this symptom has been present, and no other cause has been ascertained, removal of the calcareous masses has been followed by disappearance of the hæmaturia.

"One may, therefore, I think, be justified in suggesting some relation of cause and effect between the glands and this symptom. The close anatomical relation of the calcareous glands to the ureter is undoubted. In several cases of calcareous glands I have found the passage of the catheter up the ureter has been arrested at the level of the glands, and although other more rigid or smaller catheters pass on, I have gained the impression that some pressure or drag existed at this part.

"Looking back on cases of obscure hæmaturia, I can recall, and I think most urologists must have seen, cases in which the only cause of the hæmaturia was some condition outside the ureter, such as an appendix abscess. One cannot avoid the conclusion, therefore, that calcareous glands may, by pressure or by dragging on the ureter, be the cause of hæmaturia."

### DIAGNOSIS.

RISLEY<sup>1</sup> states that in 65 cases at the Massachusetts General Hospital, where tuberculous glands were found at operation and not diagnosed before a post-mortem, clinical records showed nothing whereby diagnosis could have been made. Many had obsolete or quiescent tuberculous glands without symptoms. Palpable masses of glands are the only diagnostic sign.

CARSON<sup>2</sup> thinks few conditions could cause similar symptoms, i.e., in uncomplicated cases; he thinks a chronic appendicitis might do it, but the pain would be less frequent, last longer, and might be localized; also more tenderness in appendix. Rectal examination: may feel acute appendix, stone in ureter (frequent micturition, X-ray, hæmaturia). Digestive disorders, intestinal parasites, and tuberculosis.

CORNER<sup>3</sup> says von Pirquet's test for bovine and human tubercle bacilli is usually negative. He thinks some infections due to bovine and some to human, some to both.

CAIRD<sup>9</sup> says:—

"A correct diagnosis was not always made. Some cases with a relatively short history were not discriminated from subacute appendicitis; in others, the mimicry of malignant disease was so perfect that the doubt was only solved on histological examination of the specimen after operation."

HOLLENBACH<sup>10</sup> considers diagnosis very difficult; the most varied pictures of organic disease may be simulated according to the position of the lymphomata.

OPIE<sup>11</sup> relies upon X-ray diagnosis.

A. T. JONES<sup>12</sup> says:—

"The diagnosis of this condition is usually not made before operation, as there can be no distinctive symptoms due to the glandular enlargement *per se*."

### RESEMBLANCE TO AND SIMULATION OF APPENDICITIS.

JOHN FRASER<sup>13</sup>, under the heading of 'Ileo-cæcal Lymphadenitis', says:—

"There is frequently a preceding attack of diarrhoea or intestinal disturbance, and the association of this is important, because the resulting alteration of the intestinal flora is possibly the exciting feature of the process. Severe abdominal pain appears—there may be vomiting, though such is by no means constant; there is considerable general disturbance and fever. The symptoms rarely last for more than twenty-four to forty-eight hours, when they abate with characteristic suddenness. How is one to distinguish such a case-history from that of appendicitis? I confess it is often exceedingly difficult—sometimes it is impossible. One or two

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features may be suggestive. In lymphadenitis the pain is local from its commencement; it is never referred. This is a point, however, which in young children it may be impossible to verify. The tongue usually remains clean, the attack subsides with characteristic rapidity; fever introduces the illness, while in appendicitis temperature is comparatively late in its appearance. It may be possible to palpate the enlarged tender glands. But I fully realize that the points I have mentioned, while difficult to verify, are capable of considerable variation, and, if your experience tallies with mine, the diagnosis of the great majority of these cases will be made after they have been operated on for what was considered to be acute appendicitis. In view of the danger in children of the 'wait and see' policy where appendicitis is concerned, it is better that things should be so."

HOLLENBACH<sup>10</sup> states that sometimes acute and sometimes chronic appendicitis is simulated, both as regards history and condition. He says:—

"During the last few years we have observed six cases, of which four simulated acute and two chronic appendicitis. Without exception these were youthful patients. The chronic cases presented a true picture of a chronic appendicitis, both as regards history (as given by the patient: anamnesis) and condition. In acute cases the general health of the patient was proportionately little disturbed, with a temperature of 40°. There was great tenderness in the cæcal region on palpation, but no definite tension of the abdominal walls, and the pulse-rate was only slightly increased. It follows from this that the height of the temperature contrasts somewhat with the general and localized condition of the patient, and this is an observation which may be eventually taken into consideration when making an alternative diagnosis. Laparotomy revealed extensively swollen lymphatic glands in the ileocæcal mesentery, which in isolated glands attained the size of a walnut. Next to the calcified glands we found, in the case of acute cases, new swellings. The appendix had undergone no noteworthy changes—certainly not to the extent that it could be considered as the cause of the affection."

SCHMIDEN<sup>11</sup> discusses the picture of tuberculous disease of the mesenteric glands in the ileocæcal angle, and describes it as typical, the knowledge of which is due to operative autopsies. In the large majority of cases it runs its course with symptoms which simulate chronic appendicitis. However, the alternative diagnosis of renal calculus, urinary calculus, gall-stones, or gynaecological trouble often has to be considered. Not infrequently, it causes severe attacks of an inflammatory nature accompanied by abdominal tension; at other times, again, symptoms appear which remind one of ileus. These misleading symptoms, accompanied by the periduenitis signs, are to be attributed to intestinal spasms.

OURSEN<sup>12</sup> mentions that in his book, *Clinical Symptomatology of Intestinal Diseases*, 1917, he has called attention to the fact (in several places) that tuberculosis of the mesenteric lymph glands may simulate acute as well as chronic appendicitis. He has shown the various points in the differential diagnoses, and has also discussed the fact that such a form of tuberculosis of the glands, just as in the cases of appendicitis, does not result in pain in the ileocæcal region but exclusively in the pit of the stomach, and therefore may simulate an appendicitis in this direction also. That such a form of tuberculosis in the ileocæcal angle may be recognized even before making an incision had once more been proved by a patient under his care, whom he handed over to a surgeon with the diagnosis of tuberculosis of the mesenteric lymph glands. The surgeon, when performing a laparotomy on this case immediately afterwards, found an acute tuberculous peritonitis, which had its origin in tuberculosis of mesenteric glands in the cæcal region.

ISRAËL has reported eight cases of tuberculous mesenteric glands, six of which came to the operating table with a diagnosis of appendicitis.

KEPPLER and ERKES<sup>13</sup> say:—

"The symptoms picture of the cases diagnosed as appendicitis is as follows: The sudden commencement of vomiting and high temperature, severe pain with contraction or tension of muscles in the right side of the abdomen, as well as constipation. Thus there can be no doubt with regard to the diagnosis prior to operation. This accounts for the fact that in only one of our cases the use of the X rays was

resorted to. Tuberculous mesenteric glands may simulate the most varied abdominal affections. For instance, the caseous and calcified glands situated in the ileocaecal angle not infrequently simulate appendicitis; those situated in the mesentery of the small intestine and in the transverse mesocolon occasionally simulate affections of the stomach or duodenum."

A. T. JONES<sup>12</sup> says:—

"Many of these cases have been operated upon with the diagnosis of appendicitis or some acute intestinal condition which presented a decidedly surgical appearance. Upon opening the abdomen, possibly a rather harmless appendix is found which is apparently not enough to account for symptoms. Further exploration in these cases reveals the mesentery studded with enlarged glands in various degrees of inflammation, and varying in size from that of a pea to that of a filbert. In some cases we may find large retroperitoneal masses composed of several glands, the whole mass being from the size of an egg to that of an orange or even larger."

BARRY and FENTON<sup>17</sup> say:—

"To surgeons who operate not infrequently for appendicitis, we feel sure in many cases it has happened they have cut down on appendices which they have been surprised to find to all appearance normal, or so little affected as to be obviously insufficient to give rise to the symptoms complained of. It is in such cases most searching investigation should be made for signs of tuberculosis in the caecum, the small intestine, and their mesenteries; should such conditions be found, an excision of the affected gut and its mesentery should be forthwith carried out. Even with an obviously diseased appendix, the surgeon should still prosecute a careful search for enlarged glands in the mesentery, and consider well the possible cause of the disease present, and how far simple appendectomy will really cure the patient; for it is in tubercular affections that the one hope of a successful issue is to deal radically with the disease whilst in its earliest stages."

MACLAREN,<sup>18</sup> speaking of unsatisfactory appendicectomies, says:—

"Deaver, in his book on appendicitis, gives the impression that it is easy to make a differential diagnosis between chronic appendicitis and the neurasthenie, even when there has been no preceding acute attack. In Deaver's fourth edition, 1913, under the heading 'Recurrent Appendicitis', he says: 'The diagnosis of chronic appendicitis is usually clear. The plainer cases give a history of acute pain referred to the region of the appendix, and examination reveals definite soreness, and upon palpation over this region even in the absence of an acute attack a diagnosis may be made with almost invariable accuracy on this combination alone, i.e., pain and tenderness in the right inguinal region'." MacLaren thinks there is a real appendicitis and one he calls the neurasthenic variety. He says:—

"They, as a rule, are young women or girls from 12 to 24 years of age (occasionally in older women, rarely in men) who are not in good physical condition, have lost from ten to fifteen pounds in weight, are worrying over family or financial troubles, or over examinations at school; they are of the flat-chested scaphoid abdominal type, and when it is necessary for them to stand a great deal they are flat-footed. They have pain in the head, back, pelvis, or abdomen, due to their general debility; they complain of tenderness on pressure in various parts of the abdomen; many of them complain of this tenderness mostly in the right inguinal region, and these are the ones who come to us for operation. But just as large a number come to us on account of abdominal discomforts after their appendices have been removed. These cases are frequently temporarily relieved, but in a few weeks their old symptoms recur. It has seemed to us that these neurasthenic cases are improved by our unintentional exercise upon them of the faith cure. For if we honestly believe that we have removed the cause of their distress, we do unconsciously exercise this power, whether we believe that we do or not. It has seemed to us that this pain and tenderness in the right inguinal region, in these cases, was usually due to the stretching of the duodenum from its fixed point under the liver to the pylorus in the pelvis. There is seldom any rise in temperature; the pulse is normal as a rule; occasionally in the younger patients it is rapid from excitement, probably due to the examination. There is no true abdominal rigidity,



and the pain in the abdomen in the chronic cases will be lessened or will disappear when the patient lies down. This last point has seemed to us to be perhaps the most important point in making a differential diagnosis between the true and the false appendicitis." He does not suggest that any are due to tuberculous glands!

## DIFFERENTIAL DIAGNOSIS OF CALCIFIED MESENTERIC GLANDS BY RADIOLOGICAL EXAMINATION.

BY L. A. ROWDEN, LEEDS.

On radiological examination of the abdomen one frequently finds shadows in the lumbar or inguinal regions; these shadows are usually smaller than a threepenny piece, but occasionally they may be much larger. They are usually multiple, and seem to be somewhat more frequent on the right of the spine. Their outline is almost always irregular and indistinct, and their appearance mottled; by palpation they can be freely moved, and, when multiple, their relative positions can be altered. When viewed from behind they appear larger than when viewed from the front. On deep inspiration they move downwards for some distance and then take a marked inward direction towards the spine. During respiration their relative positions often change. Shadows conforming to the above characteristics are caused by calcified mesenteric glands. (*Fig. 281.*)

Renal or ureteral calculi may cause shadows in these regions, and the differential diagnosis may, in some cases, not be easy. The following points of difference should be noted: (1) Renal calculus is usually single, only about 10 per cent being multiple. (2) Their shadows are usually at a higher level. (3) They move downwards and slightly outwards on deep inspiration, and do not turn inwards towards the spine. (4) They cannot be freely moved by palpation. (5) They appear smaller when viewed from the back than from the front. (6) When multiple they keep their relative positions on respiration or palpation. (7) The shadows of urinary calculi have generally a sharply defined, regular outline, and are homogeneous.

Ureteric calculi are generally single, and lie within a line joining the tips of the transverse processes of the lower three lumbar vertebræ. They cannot be moved by palpation, and do not move on respiration unless high up.

Pyelography and the opaque ureteric catheter may be of considerable help in some of the more difficult cases. Phleboliths, and the teeth or bone of dermoid cysts, are usually well down in the pelvis, and need hardly be considered in the differential diagnosis.

Radiography alone is not sufficient in many cases for the correct interpretation of the shadows. Radioscopy is essential, and, if the foregoing points are carefully looked for, a correct diagnosis is easy in most cases.

## MACROSCOPIC PATHOLOGY.

With regard to the macroscopic pathology of the condition, as seen by the surgeon, there are at least five varieties.

*Stage I:* the stage of infection, analogous to the stage of simple adenitis, e.g., cervical, which subsequently becomes tuberculous, evidenced by an



FIG. 281.—X-ray photographs by Dr. Leo Rowden.  
*The lower, right-hand picture is transposed, and represents the right iliac fossa, not the left.*

enlargement and a dull 'liver' redness of the gland or glands. This is the stage of simple adenitis, simple in that there is no naked-eye appearance of the tubercle.

*Stage 2*: the stage of 'spotted caseation', where tiny yellow spots, two, three, or four, appear in the reddened gland (*Fig. 282*). With this change there is frequently an extension of the inflammatory area from the gland to

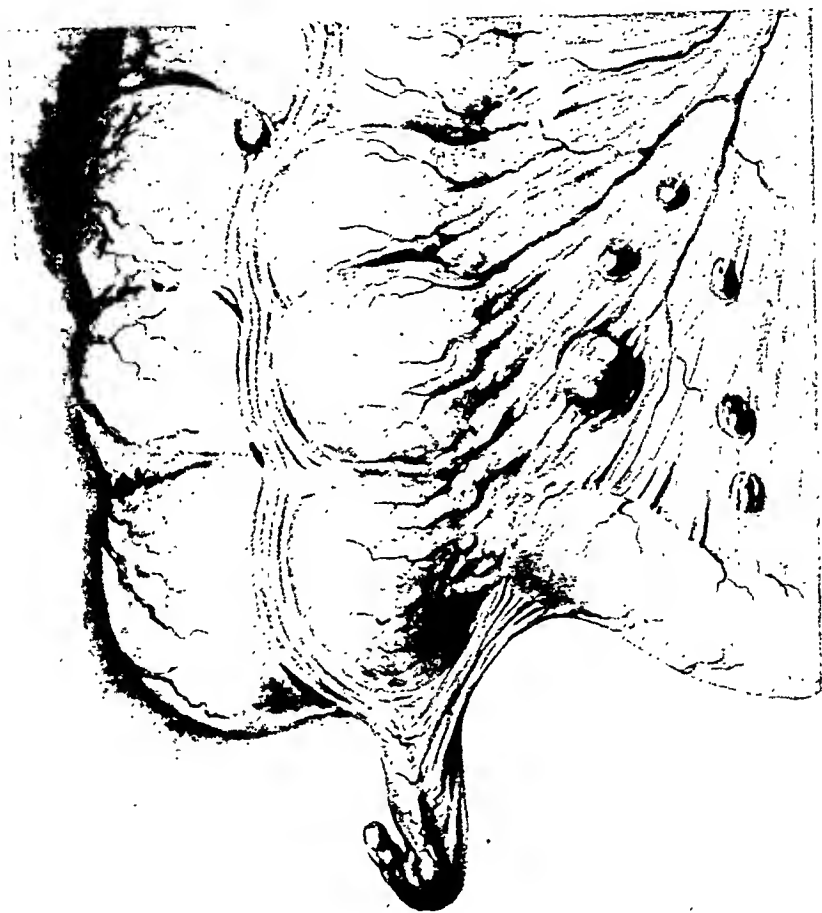


PLATE 17

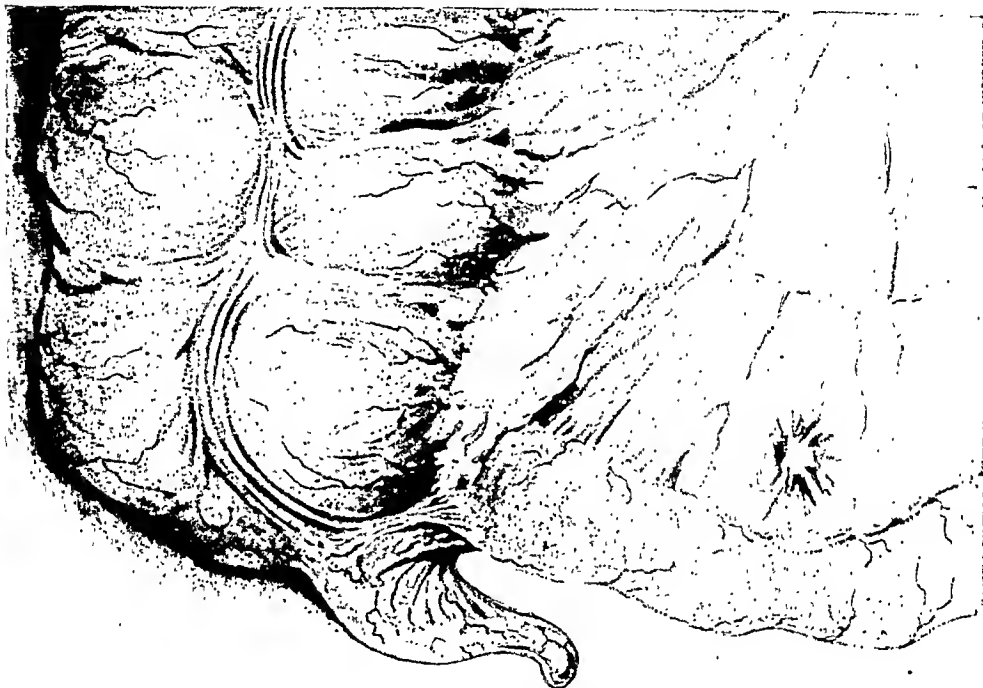
FIG. 282.—*Stage 2*. Spotted caseation of glands.

the overlying peritoneum; assuming the disease is stayed, the yellow areas may become white (calcified) and give rise to

*Stage 3*: the stage of spotted calcification. In this stage there is more local irritation, due to actual irritation of the overlying peritoneum from spicules of calcified material, which may actually penetrate it. If this happens, each spicule becomes tipped by red flagellæ (organized lymph), and the base

of each spike where it penetrates the peritoneum has its own areola of redness. It is perfectly obvious how irritative a focus this must be, and how likely to cause localized pain. This stage is associated with exudation of fluid in small quantity into the peritoneal cavity.

*Stage 4* may be termed the stage of massive caseation, where the whole gland becomes yellow. There is less liability to irritation of the overlying peritoneum, because the surface is smooth, with a string of vessels running in from borders. This type of gland may become as big as a Tangerine orange, and indeed in process of time tends to become pedunculated. It may



E. M. N. 1867

FIG. 283.—*Stage 5*. Shows a single cicatrized calcified gland, with surrounding red areola, and spasm of contiguous intestine.

even separate completely and be found lying loose in the abdominal cavity. As a rule a big caseous gland is solitary and gives rise to few symptoms. Whereas the contents of a calcified gland are sterile, an occasional tubercle bacillus is to be found in the caseous one.

By a process of further defensive change, massive calcification (*Stage 5*) may occur, but the resulting mass tends to cicatrize more than a caseous gland; it is therefore a small, irregular, intensely hard nodule with a much reddened areola flaked by red masses of lymph (*Fig. 283*). By reason of its physical characters it gives rise to a very serious degree of pain. Complicated

adhesions of omentum and intestine may give rise to large tumour formation and to serious intestinal obstruction.

### TREATMENT.

As will be indicated, there are very varied opinions as to the proper means to be taken to achieve a cure.

Those cases in *young children* where an operation for acute appendicitis shows the source of the trouble to be an acute and possibly a non-tuberculous adenitis, and not an obvious appendicitis, are treated by appendicectomy alone. Where a *mass* of caseous or calcified glands is found, removal of the appendix and closure of the abdomen is indicated, with general treatment to follow. In young children where one or two glands only are seen to be the seat of caseation or calcification, one may consider removal of the diseased glands and of the appendix. One rule should be always borne in mind, i.e., never attempt removal of a diseased gland if it lies close to the mesenteric border of small or large intestine—a faecal fistula may be the result. It would appear to be a dangerous thing to interfere in the way of either curettage of a gland or enucleation of it in the presence of an acute infection as indicated by a high temperature, though the presence of a localized peritonitis as indicated by a reddening of the overlying peritoneum should be no bar to its extirpation in the absence of severe general symptoms.

It should be borne in mind that there is a natural cure of the disease, and that such a cure will take place under favourable conditions in almost every case, though it may be a long time and it may be attended by attacks of pain. Appendicectomy should be done if the abdomen is opened at all, because, if it is not done, every attack of pain will give rise to alarming surmises that appendicitis is present.

If a diagnosis can be definitely made—usually by an X-ray examination showing calcified or caseous glands—operative interference may be inadvisable by reason of the large number of glands involved.

Where one or two calcified glands occur in adults and cause local symptoms, they should certainly be removed.

Removal of the affected gland or glands is easy so long as the gland is completely calcified or caseated, and so long as calcification has not produced rocky masses which, jutting out, entangle mesenteric blood-vessels. In the latter case enucleation may be very difficult. In a simple case, the left forefinger supporting the gland from behind, an incision is made at right angles to the line of the intestine through the peritoneum overlying the gland, by means of a dissector; the peritoneum is lifted from the front, and, in the case of the small intestine mesentery, from the back of the gland. After removal of the gland and careful ligation of every bleeding point, the peritoneum is closed by a row of Lembert sutures.

It is very often a difficult matter to remove a gland where the surface has merely become roughened by calcification—the overlying peritoneum cannot be stripped easily; in this case a tedious dissection may be necessary in order to avoid wounding vessels. It is often an easy matter to strip the upper surface, and, pushing the gland forwards with the left forefinger behind it so

as to make it pedunculated, place a pair of pressure forceps behind it in such a way as to catch the vessels running into it before cutting the gland away.

It sometimes happens that there is such a mass of inflammatory tissue in and around the capsule of the gland that excision cannot be done without a wide excision of mesentery, endangering the blood-supply of the bowel. In these cases one of two things may be done: (1) Opening and curettage of the gland, with careful hæmostasis and closure; (2) Sequestration of the gland by an omental flap or, better, an omental graft. It should always be borne in mind that the symptoms in the adult variety are due to a localized and probably only a traumatic peritonitis, and therefore a barrier of tissue between the mobile intestines and the irritating focus is all that is necessary.

As has been already said, active tubercle bacilli may be found even in completely caseous glands, whereas in completely calcified ones there are none. It would be obvious that, whatever local treatment be adopted, care should be taken to avoid infection as far as possible of neighbouring tissues, by packing and so forth. Injection of the glands with antiseptic fluid before opening them has been suggested by some authorities. 'Sterilization' of the gland cavities after curettage, by flavine and other fluids, has been largely done by the writer, and no case of exacerbation of symptoms has been encountered.

As in tuberculous peritonitis, opening of the abdomen alone seems to exert a beneficial effect, whether as a result of the congestion of the abdominal cavity and particularly of the portion handled, or as a result of the filling of the abdominal cavity with air, or, in acute cases, of the short temporary paralysis of the ileum and cæcum and the temporary fixation of the abdominal walls.

Wide excision of the cæcum with a portion of the small intestine has never been called for in the writer's experience, except where secondary involvement of bowel wall has occurred.

Whatever local treatment is adopted, it should be followed by *heliotherapy*, as advocated by Rollier at Leysin and by Sir Henry Gauvain in this country. By heliotherapy is understood treatment embracing not only exposure of the whole body to sunlight and clean air, but also the ingestion of plenty of wholesome food. It has recently been claimed that an 'artificial sunlight' produced by mercury vapour, tungsten arc, and other 'lights' which produce a special violet ray, is a very good substitute for the real thing. Rollier<sup>19</sup> gives an interesting historical account of treatment by heliotherapy.

Whatever form of tuberculosis be encountered, the question of *tuberculin* arises. In the cæcal glandular tuberculosis, tuberculin has not been shown to have any value. It is clear that in those cases where the symptoms are due to the presence of the residue of an old long-killed infection, tuberculin can have no value. But even when glands are only partly calcified or caseous, and where presumably there are living tubercle bacilli present, no effect has been noted as a special result of giving tuberculin.

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## SOME CONSIDERATIONS OF THE OPERATION OF INTERNAL URETHROTOMY AND THE END-RESULTS THEREOF.

BY G. P. B. HUDDY, LONDON.

THE 109 cases of internal urethrotomy performed at the London Hospital during the years 1921 to 1924 inclusive have been reviewed. These operations were performed on 107 patients, for in two cases the stricture recurred and a second operation was performed.

**Introduction.**—During the early years of the nineteenth century there were three methods in use for the treatment of impermeable strictures: the forcing of the stricture by a conical sound; repeated application of caustic; and perineal division of the stricture. It was during this period that a new principle of treatment was evolved. It was suggested by Howship, and later by McGhie,<sup>1</sup> that the stricture might be perforated by a sharp-cutting instrument passed through a cannula. The best known instrument of this type was that introduced in 1827 by R. A. Stafford,<sup>2</sup> surgeon to the Mary-le-Bone Infirmary, who used a lancetted stilette, contained within a silver catheter. His description of the operation runs: "The armed catheter is then passed down till the point rests against the stricture, and being held securely in such position, the spring is pressed by the thumb gently and gradually. As soon as any impression is made, the lancet should be allowed to retire into its sheath, and the blunt point of the catheter urged cautiously forward. If it do not pass on, the lancet may be again used as before." He was able to state in 1833 that he had performed this operation on forty to fifty occasions and there were no failures. Also there had been no instances of false passage, extravasation of urine, or hæmorrhage.

For the development of the modern operation of internal urethrotomy we are largely indebted to the French surgeons, who substituted a clean incision of the stricture for the brutal and non-surgical method of divulsion.

The early types of urethrotome incised the stricture from behind forwards, the most famous of these being the Civiale instrument. In order to use this latter it is necessary that the stricture should admit a F. 8 bougie. Civiale's urethrotome was strongly advocated by that great authority, Sir Henry Thompson, who pointed out that if preceded by preliminary continuous dilatation it could be used for even the smallest of strictures.

The year 1855 saw the introduction of the famous Maisonneuve urethrotome, which has since been modified by Teevan and Sir John Thomson-Walker. Since that date innumerable other types and modifications have been introduced both by surgeons and by instrument makers, but their popularity has been short-lived, and the modified Maisonneuve is the one most commonly in use to-day. This instrument was employed for the series of cases under consideration.



An important advance was made by Otis,<sup>3</sup> who, in 1874, devised an instrument for measuring the urethra—a urethrometer, by means of which he showed that the lumen of the urethra was much wider than had been supposed. Previously it was held that if a French 21 bougie could be passed the urethra could not be considered to be strictured; but he showed that the average urethra passed with ease a French 32.

**Indications for and Contra-indications against Operation.—**

*Indications.*—The indications for the use of internal urethrotomy depend upon the views of various surgeons. The main types of stricture which require urethrotomy are:—

1. The very narrow stricture which is only permeable with considerable difficulty and in spite of repeated efforts will not dilate readily.
2. The resilient stricture which dilates but as readily contracts.
3. The stricture in association with a perineal fistula, provided the fistula persists solely because of the presence of a stricture in front of it and not on account of a chronic infective condition.

*Absolute Contra-indication.*—Impermeability.

*Contra-indications to Immediate Internal Urethrotomy* (i.e., indications for the two-stage operation).—(1) Marked urinary infection; (2) Renal insufficiency; (3) Peri-urethral abscess; (4) Extravasation of urine.

**THE TWO-STAGE OPERATION.**—The points just referred to as indications for the two-stage operation require further consideration.

1 and 2. *The Presence of Urinary Infection and Back-pressure Effects.*—These conditions usually go hand in hand. In 42 cases, none of which were fatal, albumin and pus were definitely noted as being present in the urine. Manifestly the presence of these two bodies in the urine is no absolute contra-indication to internal urethrotomy. Their presence, however, should act as a danger signal, and investigations be made as to the renal efficiency and possibly also cholesterol content of the blood. If these tests are unsatisfactory, then preliminary suprapubic cystotomy should precede internal urethrotomy, for free drainage is essential.

External urethrotomy combined with division of the stricture has been recommended by some authorities, but this is often a very tedious and time-consuming process, and so is obviously bad for such a patient. Moreover, perineal drainage results in increased fibrosis at this, the commonest, situation for a stricture.

3. *The Presence of a Peri-urethral Abscess.*—In such cases the abscess should first be drained, and when the wound is clean internal urethrotomy should be performed. This method was adopted in 5 of this series of cases, with very satisfactory results.

Peri-urethral suppuration is often held to be an indication for external division of the stricture. This would not appear to be the method of choice, because that mentioned above not only permits of free drainage, but also of a comparatively clean division of the stricture when all infection has subsided. If the abscess is opened and the stricture at the same time divided, infection is spread through the whole extent of the wound down to and including the urethral mucosa, with resulting additional fibrosis.

4. *Extravasation of Urine.*—This has been considered in a previous paper.<sup>4</sup>

The treatment recommended is primarily perineal drainage of the bladder combined with free incisions into the infected tissues, followed at a later date by internal urethrotomy.

**The Operation and its Complications.**—It is not proposed to give a full account of the operation in this paper. The method employed in every case was the ordinary Maisonneuve operation, immediately followed by dilatation of the urethra to its full calibre, and the tying in of a No. 12 catheter.

The complications met with in this series of cases are discussed below :—

1. *Urinary Fever.*—Rigors and pyrexia may occur at one or both of the following periods : (a) Immediately consequent upon the operation ; (b) Following upon removal of the catheter or dilatation.

a. Considering the first of these, rises of temperature are frequent, as will be seen from the following figures : No pyrexia, 49 ; Slight pyrexia (under  $100^{\circ}$ ), 22 ; Pyrexia (over  $100^{\circ}$ ), 28 ; Rigors, 10. The foregoing show that 38 of the 109 suffered from definite constitutional disturbance, while of the remaining 71, 49 showed no reaction whatever.

b. The second group consists of 29 cases, namely : Rigors, 12 ; Pyrexia, 17. Dilatation was a more frequent causative factor than the removal of the catheter.

R. Harrison<sup>5</sup> stated that rigors could be prevented in cases of internal urethrotomy by draining the bladder through a perineal incision. This immediately does away with some of the advantages of internal urethrotomy, for it prolongs convalescence and increases the difficulties of nursing, as urine drains through the perineal wound.

2. *Hæmorrhage.*—A certain degree of bleeding is an inevitable result of this operation, as direct means of hæmostasis cannot be applied. In no instance in this series was it of sufficient degree to cause alarm. In discussing this subject, in *Keen's Surgery*<sup>6</sup> it is stated : "The complications most often observed are extravasation . . . and, not rarely, fatal hæmorrhage from wounding of the dorsal vein, the plexus of Santorini, or the artery of the bulb." It would seem that the danger of hæmorrhage has been exaggerated, no doubt owing to the fact that on theoretical grounds it should be expected, because an incision is made into a highly vascular cavernous tissue. Sir Peter Freyer<sup>7</sup> reported several hundred cases of internal urethrotomy without a death, and very few attended by hæmorrhage of any importance. Sir John Thomson-Walker<sup>8</sup> also considered this danger exaggerated. Deaths, however, do occur from this cause. Hurry Fenwick<sup>9</sup> in 1888 recorded two fatal cases, not occurring in his own practice, and Myles,<sup>10</sup> ten years later, also recorded two fatal cases.

This harassing complication can in some measure be avoided—first, by holding the urethrotome strictly in the mid-line so that the knife enters the interval between the two corpora cavernosa, and secondly by the tying in of a catheter. If the hæmorrhage is profuse, perineal pressure combined with the use of an in-dwelling catheter will usually suffice. The degree of distention of the bladder must be carefully watched, for as a result of the perineal pressure the blood, instead of appearing at the meatus, may pour into the bladder. If the bleeding still persists, drastic treatment must be adopted before the patient's condition is hopeless. External urethrotomy must be

performed at the site of the incised stricture, a petticoated catheter passed, and the wound packed firmly around it.

3. *Hemiplegia* (right).—This occurred in one case on the 4th day following the operation. A similar complication arising on the 6th day was recorded by the Marine Hospital Service, U.S.<sup>11</sup> following division of a stricture by the Otis urethrotome; the cause was found to be a hæmorrhage into the left corpus striatum.

4. *Atonic Bladder*.—One case occurred, which rapidly cleared up with regular catheterization. This, strictly speaking, is a complication of the urethral stricture rather than of the operation.

5. *Peri-urethral Abscess*.—This ensued in a case in which no in-dwelling catheter was used. The abscess was incised, and a second urethrotomy was required later.

6. *Epididymo-orchitis*.—Two cases occurred, in both of which a catheter had been tied in, for two days and four days respectively.

There was no case of urinary extravasation.

**After-treatment.**—Before discussing the results a brief indication as to after-treatment must be given, as the results are so entirely dependent upon this. As will be seen in a later paragraph, it is advisable to allow the catheter to remain in position at least four days. Four days after its removal the first dilatation is performed, up to at least E. 14/16, and in some cases even to E. 16/18. Provided no pyrexia follows, the patient may be allowed up, and discharged twenty-four hours later. Regular dilatation up to the sizes already mentioned must now be pursued.

The following table will serve as a guide to the regulation of the after-treatment :—

INTERVALS			NO. OF ATTENDANCES	
1 week	..	..	..	4
2 weeks	..	..	..	3
3 "	..	..	..	3
4 "	..	..	..	3
6 "	..	..	..	2
8 "	..	..	..	1
10 "	..	..	..	1
12 "	..	..	..	1

Each interval is then increased by a month until the patient is seen twice yearly. In favourable cases the interval may later be increased to nine months or even a year, but the patient must always be kept under observation. The above is a guide only to be used for an average case, and must be modified as required for each individual.

Kollmann's dilator is a valuable instrument to use when the meatus is narrow or when over-dilatation of the stricture is desired.

For the early dilatations novocain should be used, for instrumentation is painful, and this is an important factor in influencing the regular attendance of the patient. Its use may be continued or not at the discretion of the surgeon.

**The Post-operative Use of an In-dwelling Catheter.**—This, which is an important feature of the after-treatment, has already been mentioned, and deserves further consideration.

The value of this procedure is denied by some surgeons (Coulson<sup>12</sup> and Sir Peter Freyer<sup>7</sup>). It has, however, the following advantages:—

1. By bringing a certain amount of pressure over the incised area it diminishes bleeding.

2. It conducts the urine away from the bladder, and as this urine is often infected, the risks of infection of the urethral wound and extravasation are thus considerably diminished.

3. If unsupported, the sides of the wound tend to fall together and the stricture tends to be re-formed (*Fig. 284*).

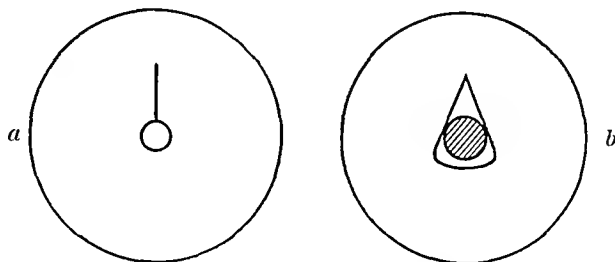


FIG. 284.—Showing incised stricture (*a*) without catheter, (*b*) with catheter inserted.

4. If no catheter is used, each dilatation must result in the tearing of young fibrous tissue binding the edges of the cleft, and so cause increased fibrosis.

In reviewing the 109 cases, there is no evidence of its having produced any ill effects.

*Period during which Catheter should Remain in Position.*—In comparing the post-operative pyrexia, etc., the following table was evolved, three groups of cases being chosen for the purpose.

PERIOD OF CATHETER USE	TOTAL CASES	NO PYREXIA	POST-OPERATIVE RIGORS	POST-OPERATIVE PYREXIA
No catheter	10	5	1	4
2 days	41	17	3	21
4 „	12	5	2	5
Totals	63	27	6	30

An attempt was made to estimate the end-result of these same cases, and the 4-day cases showed the best results; but the figures obtained do not convey an accurate impression, because some patients attended for dilatation while others failed to do so. In considering whether the duration of the in-dwelling catheter may be of importance, the following points should be noted:—

1. One patient, mentioned below, in whom the catheter remained only two days, is attending the Stricture Department, and it is only with extreme difficulty that a 12/14 English bougie can be passed.

2. The process of healing commences early, as is shown by the frequency with which pyrexia and rigors follow the re-opening of the wound

by dilatation. A catheter, so long as it remains in position, prevents the cementing together of the raw edges by this rapid healing, and provided it causes no ill effects should be retained at least four days.

3. If the process of healing just mentioned occurs with unusual rapidity, it may result in rapid recurrence of the stricture. There was one such case in this series, in which a second urethrotomy was necessary a few weeks later. The catheter had remained in only two days. A somewhat similar case is that of the man, mentioned above, who developed a peri-urethral abscess and later required a second internal urethrotomy. It will be observed that an in-dwelling catheter had not been employed.

### RESULTS OF OPERATION.

**Mortality.**—Two patients died following the operation. In the first of these, carcinoma of the bladder, vesical calculus, and purulent nephritis were associated with the urethral stricture. This case obviously can hardly be considered in estimating the immediate mortality. One case remains in which the death was consequent upon the operation :—

A male, age 52, was admitted with a long history of retention of urine with overflow. Internal urethrotomy was performed and a catheter tied in for four days. Irregular pyrexia ensued, and one rigor followed the dilatation. Death occurred on the twelfth day. At necropsy the bladder was dilated, hypertrophied, and inflamed: the kidneys were hydronephrotic and showed a condition of pyelonephritis.

This case serves to emphasize the fact already mentioned, that where there is any doubt as to the renal efficiency this should be investigated and if necessary a two-stage operation be performed.

In a series of 1018 cases of internal urethrotomy at St. Peter's Hospital there were 8 deaths.<sup>13</sup> It will be observed that the mortality is roughly 1 per cent.

In all the cases reviewed in this paper the operation was a life-saving one, in so far as the patient was relieved of the prospect of the severe back-pressure effects of a very narrow stricture which resisted dilatation, and was not an 'opération de complaisance'. If the operation is performed on strictures of a wider calibre, the operative risk is obviously much less, for the likelihood of kidney failure or urinary infection is much diminished. This is shown in the teachings of Otis<sup>3</sup> in 1881, for he states regarding indications: "In my experience, the greatest number of strictures which call for operative measures are the so-called strictures of large calibre which produce and prolong urethral discharge". He then gives a total of 2163 operations by various surgeons without a death or permanent disability of any sort.

These results are so satisfactory, and especially so in the case of a wide-bore stricture, that the scope of internal urethrotomy should be enlarged, and it should be carried out in those cases which are only dilated with difficulty and by heroic persistence on the part of the patient and surgeon.

**Late Results.**—Subtracting the 2 deaths, 105 patients remain. These fall into the following classes: Died since discharge from hospital, 8; Traced, 78; Not traced, 19.

The 8 deaths all occurred in patients who failed to attend the Stricture

Department. *Cause of death*: Recurrence of stricture and kidney failure, 2; Cystitis and pyelonephritis, 1; Extraneous causes, 3; Cause not determined, 2.

The two cases of recurrence are very disappointing, for this is definitely preventable. It serves to emphasize the fact that internal urethrotomy of itself cannot be regarded as a curative operation. Scar tissue remaining must always be a potential cause of stricture, and the importance of regular post-operative dilatation cannot be too strongly impressed upon the patient and even the doctor. Following an internal urethrotomy, the urethra is usually enlarged even if after-treatment is not pursued, but in a few cases the additional fibrosis caused by division of the stricture results in further constriction.

Of the remaining 97 patients 78 have been traced, and the results are entirely satisfactory and the patients most grateful, many having expressed their thanks in eulogistic terms.

Of these 78, 44 are still under observation at the Stricture Section of the Genito-urinary Department, and have attended regularly. In this group the results can only be described as brilliant. All, with two exceptions, admit readily Thompson's sounds of the size English 14/16 or over; the intervals of dilatation have been prolonged up to many months, and in a few cases as much as one year. Of the two exceptions, the first is a patient who at operation was only dilated to E. 12/14 and now admits E. 13/15; the second is a man with a very dense tunnel stricture which can only be dilated to E. 12/14 with difficulty.

Information has been obtained from 34 who for different reasons are not attending the Department, some living at a considerable distance and attending their own doctors, a small number being afraid of dilatation. These patients, without exception, have expressed their complete satisfaction at the result. In 3 patients only has there been any recurrence of dysuria, but 9 others, although they state definitely that there is no difficulty of micturition, reply to another question that the stream is not quite as free as on discharge from hospital.

These results can be summarized thus:—

Attending the Stricture Clinic—results brilliant	..	..	..	44
Not attending the Stricture Clinic—quite well	..	..	..	22
“ “ “ “ “ —some dysuria	..	..	..	3
“ “ “ “ “ —no dysuria but flow not quite so free	..	..	..	9
				—
				78

Of the patients in the last two groups, all but one have failed to pursue regular instrumentation. Once again, therefore, the great post-operative essential is regular dilatation.

### CONCLUSIONS.

1. The operation should be preceded by suprapubic drainage of the bladder if there is evidence of renal insufficiency or marked urinary infection.
2. A peri-urethral abscess should first be drained, and internal urethrotomy performed when the infection has subsided.

3. Urinary extravasation is best treated by free incision into the infected parts combined with perineal drainage of the bladder, internal urethrotomy being performed at a subsequent operation.

4. The dangers of hæmorrhage have been exaggerated.

5. The results are extremely satisfactory, and the operation should be carried out in any case where dilatation is difficult or unsatisfactory.

6. An in-dwelling catheter has beneficial results, and should, unless any complication arises, remain in position four days.

7. Post-operative dilatation is essential.

It is with the greatest pleasure I record my thanks to Mr. Hugh Lett, Director of the Genito-urinary Department, and Mr. G. E. Neligan for permission to use their case records, and for their kindly and ever stimulating interest and advice in the work of the Stricture Section of the London Hospital Genito-urinary Department.

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## THE APPLIED ANATOMY AND PHYSIOLOGY OF THE THYROID APPARATUS.\*

By GEORGE SCOTT WILLIAMSON, LONDON.

(Being the Arris and Gale Lecture delivered before the Royal College of Surgeons of England, on Feb. 18 and 20, 1925.)

### INTRODUCTORY.

IN presenting some new aspects of the anatomy and physiology of the thyroid apparatus, I wish to indicate how these new facts can assist the clinician in arriving at a more rational understanding of the relation, one to another, of the groups of diseases of the thyroid gland, in the hope that a more critical study of the symptoms presented by these diseases will ultimately lead to an understanding of the part played by the thyroid gland in the metabolic turnover. The subject, I think, is one that will repay close study by team workers. There is a tendency to discredit the anatomist and to exclude him from these teams. It is forgotten, perhaps, that the design and architecture of any engine is very completely controlled by the nature of the fuel and the manner in which that fuel is to be transformed into work. An engineer presented with a piston or a cylinder, or other part—even a damaged (pathological) member—of the engine, can very accurately deduce the nature of the fuel and the method of burning it. We are only beginning again to study anatomy from such a point of view, and already this is yielding results, as may be seen in the work of Aschoff and his pupils.

The germ of this vital attitude to anatomy is manifest everywhere in the works of John Hunter. It has awaited the quickening that lies in the new ground of modern physiology, as exemplified in the remarkable books of the late Sir William Bayliss.† It has but a remote relation to the study of the effect of death on cells and tissues, and none to the classificatory treatment of pathology, as Bland-Sutton has pointed out in his treatise on tumours.‡ I propose, therefore, to look at the anatomy of the thyroid apparatus from this 'vital' point of view.

The subject matter of this paper represents the joint work of Dr. Innes Pearse and the author.

### ANATOMY.

**The Microscopic Anatomy of the Thyroid Gland.**—I will begin with a brief outline of the microscopic anatomy of the thyroid gland. The detailed studies of the minute anatomy were published by us in 1923.<sup>1</sup>

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\* From the Laboratories of the Royal College of Surgeons, England, and from the Dunn Laboratory, St. Bartholomew's Hospital.

† *Principles of General Physiology; Interfacial Forces and Phenomena in Physiology.*

‡ *Tumours, Innocent and Malignant.*



In the fœtus the epithelium of the thyroid gland is arranged in the form of long columns of tissue. They run a straight course, or are coiled and folded on themselves. Epithelial columns of this nature occur in all normal glands, in both sexes, and at all ages. In the adult gland they constitute the so-called 'interfollicular' tissue of the literature. Microscopic identification of these columns in the mass of interfollicular tissue depends upon the existence of a system of microcapillaries occupying a central position in each column.<sup>2</sup>

In certain conditions of marasmus in man, and in animals also during starvation, the whole adult thyroid epithelium may take this form. As a

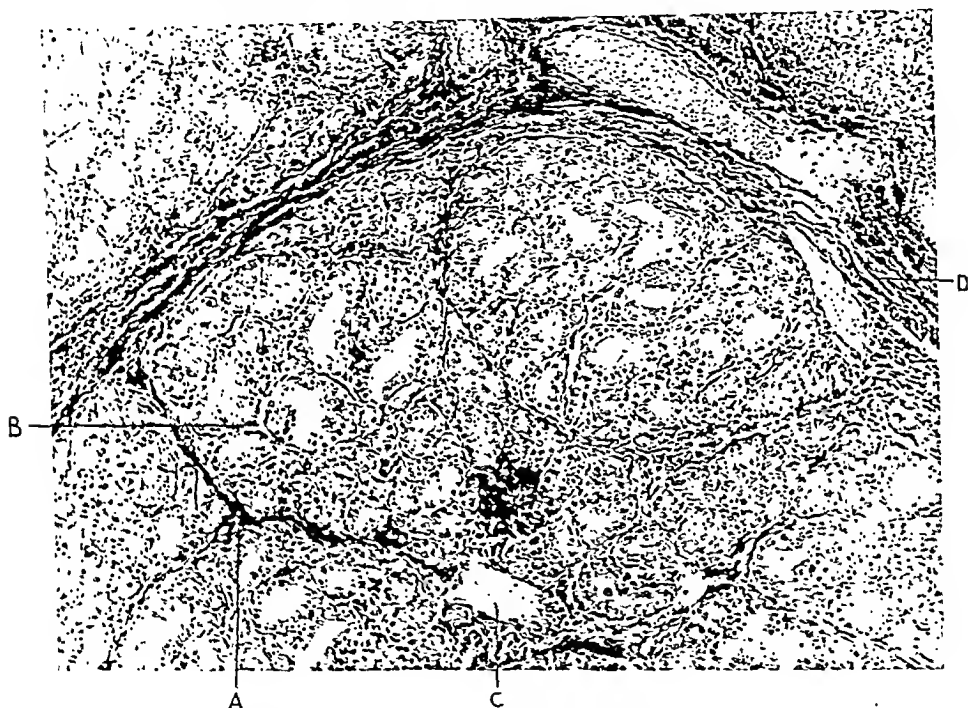


FIG. 283.—Microphotograph illustrating the gland-units within a lobule of the thyroid gland. This feature of the structure of the gland is only apparent when the tissue is engaged in producing secretion proper. The content of the follicles is not colloid. A, The thin fibro-elastic tissue defining the gland-units; B, The lymph sinusoid appearing as open inter-follicular spaces; C, The central intralobular lymph channel communicating with the lymph sinusoids; D, The interlobular fibrous-tissue stroma in which run the interlobular vessels.

rule, however, the solid core of the epithelium columns is replaced either by an accumulation of secretion on the one hand, or of colloid on the other hand. Although these events do not alter the essential structure of the columns, they do alter their appearance on section, and suggest a follicular nature for the tissue. This has led in the past to a description of the thyroid gland as being a collection of independent follicles, and it must be admitted that, were the phase of colloid storage alone studied, such a simple conception of the gland mechanism would appear to suffice. If, however, all phases of

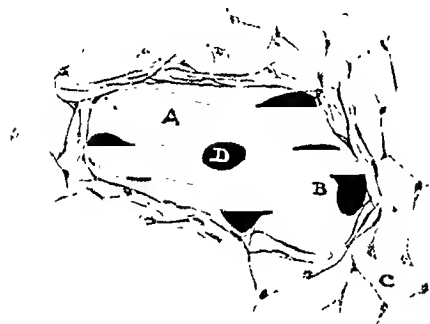
function are to be reconciled, a larger area, and a design of greater intricacy than the simple follicle, must be ascribed to the unit of function in the thyroid gland.

*The Gland-unit.*—The simplest comprehensive grouping of tissues in the thyroid gland is the 'gland-unit'. A gland-unit consists of an endothelial-lined cavity enclosing coiled columns of epithelium accompanied by a basket-work system of capillaries. These capillaries enter the endothelial sac at its hilum, where they take origin from the intralobular capillaries (*Fig. 285*). The endothelial sac itself is an extension of the perivascular lymph channels which accompany the intralobular capillaries. The sac thus encloses a lymph space.

Such an association of structures recalls the liver. The gland-unit of the liver is an endothelial-lined sac, the portal blood sinusoid, in which lie columns of liver epithelium enmeshed in a plexus of Kupffer lymph capillaries.

In the thyroid gland, as in the liver, the endothelial sac is not always

a mere potential space. Between the columns of epithelium, separating them one from another, there is often to be seen a thin, fluid non-colloid, lymph-like matter (*Fig. 285*). (See also Hürthle,<sup>3</sup> Matsunaga,<sup>4</sup> McCarrison,<sup>5</sup> and others.) Matsunaga had injected areas of this lymph sinusoid, which he calls a peri-epithelial space. Moreover, both in the human foetus and the adult, this space is frequently occupied by lymphocytes. We have been able to trace the contents of the space to the perivascular lymph channels within the lobule, and beyond this to the lymph spaces of the interlobular interstitia, and thence to the hilum of the gland. Thus the sac in which



*FIG. 286.*—Drawing from human foetal thyroid (after Norris), showing method of formation of peri-epithelial spaces into which grow the thyroid capillaries. A, Undifferentiated mass of multinucleated protoplasm—the thyroid *Anlage*; B, Peri-epithelial space; C, Interstitial cells giving origin to thyroid capillaries; D, A vacuole before rupture.

the epithelial columns and their capillaries lie is an extension of the lymph system, and may be called a lymph sinusoid.

*Embryology and Comparative Anatomy.*—We may now turn to studies in the embryology and comparative anatomy of the thyroid for further evidence in support of such a construction in the gland.

The thyroid epithelium in the embryo appears first as an undifferentiated mass of multinucleated protoplasm ('*Anlage*') (*Fig. 286*). According to the latest work of Norris,<sup>6</sup> which we can corroborate, within this mass of protoplasm there appears a number of vacuoles; Norris, following the fate of these vacuoles, discovers that they rupture on the periphery of the *Anlage*. They thus form a series of peri-epithelial spaces between the thyroid *Anlage* and the surrounding connective tissue. Into the peri-epithelial spaces so formed the special thyroid blood-vessels grow and spread themselves over the

developing epithelium. Much later, in the column-like outgrowths of epithelium accumulations of secretion give a follicular appearance to the epithelial tissue. The peri-epithelial spaces of the embryo, therefore, would seem to be the forerunners of the lymph sinoids of the fully developed thyroid gland.

Comparative anatomy affords similar evidence. We are deeply indebted to Mr. Burne, Physiological Curator, Royal College of Surgeons of England, for the favour of anticipating the publication by him of the following facts. By injecting and tracing the lymphatic system in adult angler fish (*Lophius piscatorius*), Mr. Burne has demonstrated the existence in the neck of a large ventral lymph sac\* (Fig. 287). Our interest lies in the fact that in the wall of this lymph sac is set the thyroid gland. The thyroid follicles project into the lymph sac, or into bays formed by its reticulation (Fig. 288). The follicles are often stalked, floating at anchor in the fluid contents of the sac. Furthermore, the epithelium of the follicles is in direct contact with the contents of the lymph sac, except of course where the blood capillaries may intervene (Fig. 289)

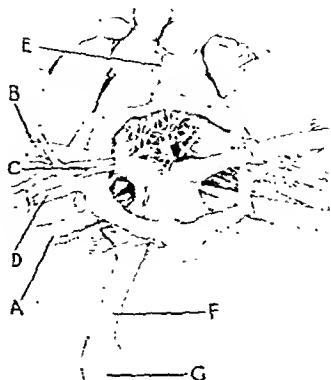


FIG. 287.—Photograph of drawing by Mr. Burne, R.C.S. Eng., of the thyroid body of adult angler fish, opened from the ventral surface, showing the central lymph sac, X, which has been opened to show the inner surface with its reticulations upon which lies the thyroid tissue. A, Hypobranchial artery; B, Afferent branchial artery II and III; C, Afferent branchial artery I; D, Lymph channel from ventral branchial sinus to the thyroid lymph sac; E, Lymph channel from ventral surface of intermandibular space; F, Lymph channel leading from thyroid sac to inferior jugular vein, G.

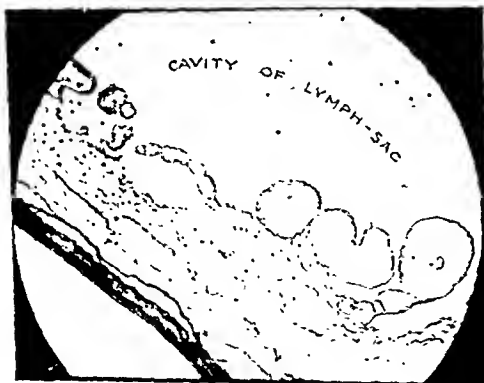
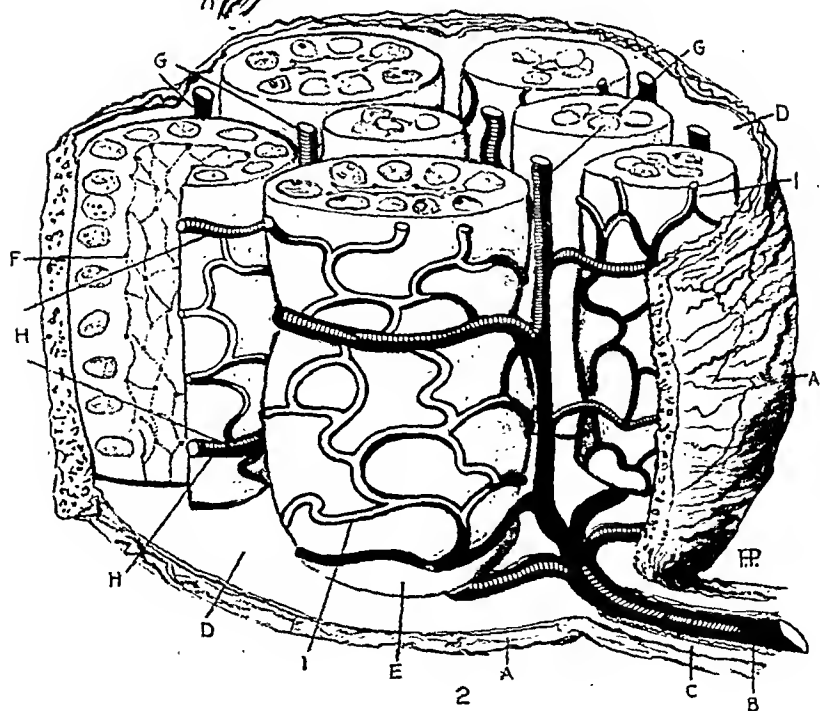
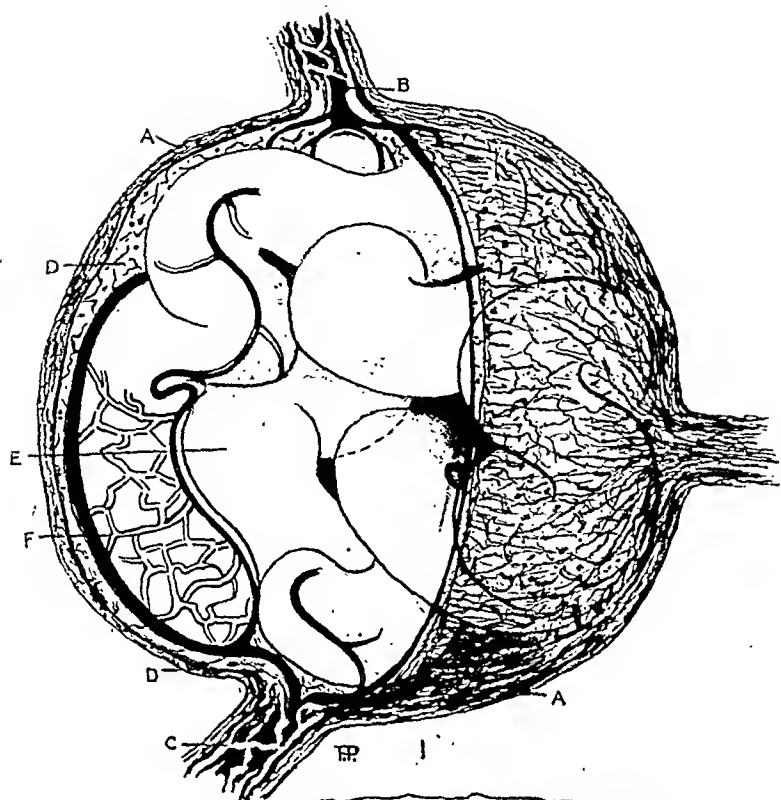


FIG. 288.—Wall of the ventral lymph sac of the adult angler fish, showing columns of thyroid tissue projecting into the lymph cavity and thus floating in the lymph itself. ( $\times 40$ ).



FIG. 289.—Wall of the lymph sac of adult angler fish, showing the naked thyroid epithelium lying in the lymph. A, Wall of sac; B, Lymph cavity. ( $\times 65$ ).

\* See Specimen No. AS50A, Museum, Royal College of Surgeons, England.



Thus embryologic and comparative morphology afford further substantiation of our concept of the thyroid gland-unit being fundamentally associated with the lymphatic system. Thus we conclude that *the morphological unit of the thyroid gland is a lymphatic sinusoid containing coiled columns of epithelium which are enmeshed in a basket-work of specialized blood capillaries* (Figs. 290, 291).

#### DESCRIPTION OF FIG. 290.

1. Diagrammatic representation of an isolated gland-unit of the thyroid organ. A, Fibro-elastic tissue capsule, partially removed to show the interior of the gland-unit; B, Intralobular blood-vessel, surrounded by C, the intralobular perivascular lymphatic plexus, which expands to become D, the serous cavity of the lymph sinusoid, in which lies E, the coiled and convoluted epithelium, enmeshed in F, the specific capillary plexus, which is an extension of the intralobular blood-vessels.

2. Schematic presentation of a section of a gland-unit, illustrating the blood-vascular arrangements. I, Anastomotic basket-work of capillaries stretched between H, the communicating capillaries which arise from G, concomitant capillaries which run in the length of E, the epithelial columns (seen in section) lying within D, the serous cavity of the lymphatic sinusoid. C, Intralobular lymphatics. B, Intralobular blood-vessels. A, Fibro-elastic tissue capsule of the gland-unit. (Diagram taken from the author's original paper in the *Journal of Pathology and Bacteriology*.)

**Structure of the Lobule.**—If the thyroid gland be constructed on the basis of the gland-unit, we find that a number of units are collected together by strands of interstitial connective tissue to form a lobule. The lobules are peculiar in that, although true blood-vessels (interlobular vessels) run in the interstitia surrounding them, all the intralobular vessels are simple endothelial vessels. They are provided with no support other than that of the fibro elastic tissue capsule of the gland-units between which they lie. *It follows therefore that, within the thyroid lobule, artery and vein are indistinguishable.*

Furthermore, within the lobules there exists a central lymph channel common to the contained gland-units (see Fig. 285). This central lymph channel has much the same relation to the lymph sinusoid of the thyroid gland as the hepatic vein has to the portal blood sinusoid in the liver.

The lobules of the gland are bounded by an ample fibrous-tissue stroma in which arteries, veins, and lymphatics run to and from the lobules and their gland-units. This

stroma knits lobule to lobule, thus forming the lobes of the gland.

**The Hilum and Suspensory Ligament.**—In each lateral lobe of the thyroid gland there is a clearly-defined hilum at which enter the arteries which supply the gland-units (see Fig. 310).

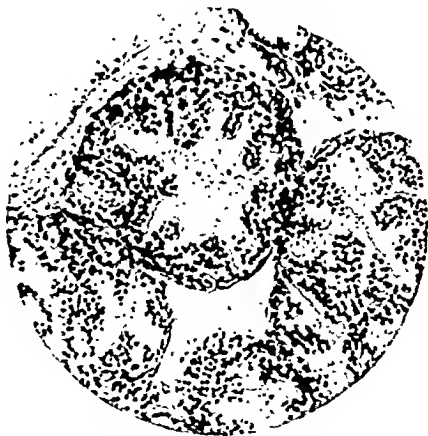


FIG. 291. — Microphotograph of the thyroid gland of a dog, taken from the work of Halsted, 1888. One gland-unit is seen surrounded by its fibro-elastic capsule. Three others partially occupy the field. All the gland-units are in the secreting stage of function. ( $\times 220$ .)

In most glands the hilum is a very clearly defined zone situated on the mesial or tracheal surface of each lateral lobe. It is set nearer the posterior

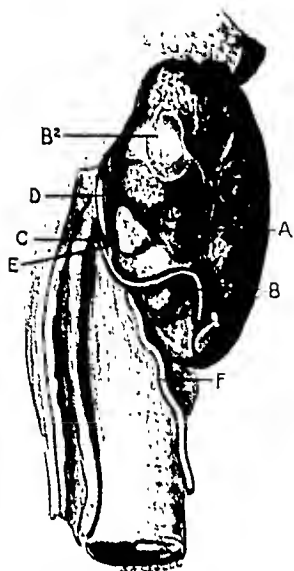


FIG. 292.—Posterior view of right lobe of human thyroid gland *in situ* (half oesophagus removed), to show the structures in the hilum of the thyroid lobe. A, Parathyroid body; B, Thymic nodes; B2, Thymic node fibrosed; C, Laryngo-oesophageal branch of inferior thyroid artery; D, Anastomotic branch from superior thyroid artery; E, Parathyroid artery; F, Recurrent laryngeal nerve.

**Thyroid Arterial Supply.**—The functional arterial supply to the gland enters each lobe at the hilum (*see Figs. 309, 310*), from which it can be traced to the capillaries. This functional arterial supply is derived from the four or five terminal branches of the inferior thyroid artery. These are joined either by an inconstant anastomotic branch, or sometimes by a laryngeal branch, of the superior thyroid artery (*Figs. 292, 310*). As a rule the anastomotic branch is insignificant; in rare cases, it constitutes the largest source of arterial blood entering the hilum. *In any case, however, the blood supplying the functional gland-units enters the lobe at the hilum.*

What, then, is the purpose, for the thyroid gland, of the superior thyroid artery, apart from its insignificant anastomotic

margin of the tracheal surface of the lobe (*Fig. 292*). At the hilum a marked condensation of fascial tissue occurs, forming a firm band extending from the hilum to the thyroid and the cricoid cartilage. This ligament was described by Berry,<sup>7</sup> and shown by him to be of the nature of a suspensory ligament for the thyroid gland. Associated with this condensed tissue are the bundles of the recurrent laryngeal nerve running to their termination in the larynx. This ligament, together with some muscular slips on the anterior surface of the thyroid lobes, ensures the movement of the gland with the larynx, while at the same time it permits considerable movement of the gland on trachea and larynx. This latter movement is liable to early interference in malignant infiltrations, and in certain specific fibroses in pathological states of the thyroid gland.



FIG. 293.—Drawing of an injection of the carotid artery in the human cadaver showing the free passage of the injection mass into the superficial thyroid venous plexus and thyroid veins. Note the insignificant branches of the superior thyroid artery passing to the fascial capsule, and the absence of any large branch of supply to the depths of the thyroid gland.

supply to the inferior thyroid artery? Injection of this vessel indicates that it supplies twigs to the interstitial-tissue septa from the surface of the gland: these are largely distributed to the anterior and lateral surfaces of the gland (*Fig. 293*). The suggestion is that the superior thyroid artery has a certain nutritive function to the interstitial tissue, recalling the distribution of the hepatic artery in the liver.

There is to be noted one other peculiarity of the vascular supply. In nine cases out of ten, attempts to inject the superior thyroid vessels in the living animal as well as in the cadaver result in the injection mass readily passing to the larger surface veins (*Fig. 293*). It fails almost completely to pass through the intimate capillaries of the gland-unit. It would appear that there is a 'shunt circuit' in the interstitial tissue of the organ—a mechanism whereby physiological necessity determines the route traversed by the blood. It is as well to recall the weighty speculative conclusions that have been drawn from the measurement of the blood-volume issuing from or entering the thyroid arteries. Such measurements may refer to blood that has never been in contact with the thyroid parenchyma. This 'shunt circuit' may explain the comparative freedom of the gland from gross infection, and may account for the anomalous result of perfusion experiments.

As to the significance of this free arterio-venous anastomosis between the thyroid arteries and the voluminous thyroid venous plexus we venture no opinion. The old anatomists suggested a blood reservoir, and Barcroft's interesting work on the spleen,<sup>8</sup> and the recent observations on plethora (Truncatek<sup>9</sup>) may revive the speculation. There is still much to learn concerning the blood-vascular system of the thyroid apparatus.

#### **The Thyro-thymic Lymph-vascular Connection.**—

Far more important is the lymph-vascular system of the thyroid apparatus. This deserves the closest study. It is a difficult subject, since complete injection of the lymphatics has not yet been achieved. Fortunately the continued activity

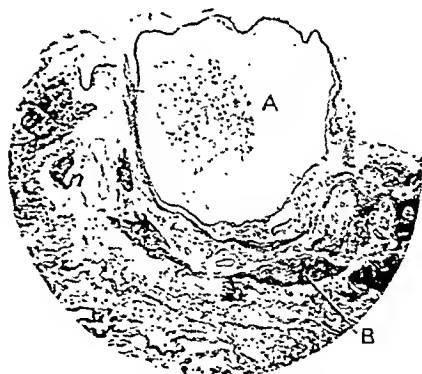


FIG. 294.—Cross-section of a dilated lymphatic channel (A) in the stalk of the adult human thymus. B, Thymic tissue. ( $\times 35$ .)

of the gland itself provides us with a means, laborious in the extreme, of tracing the continuity, if not the course, of the lymphatic system. In a previous communication we pointed out the association of congestion of lymph in the thyroid sinusoids and lymph channels with similar accumulations in the thymic tissue and parathyroid bodies. This lymph has a tendency to accumulate in localized dilatations, or pseudo-cysts, occurring most frequently in the region of the hilum. Surgeons and embryologists are familiar with these endothelial-lined pseudo-cysts, which are so easily distinguished from colloid cysts by their steel-grey glistening endothelial membrane, and in

general by the fluidity of their contents (*Fig. 294*). Quite commonly, a series of cysts of this description can be followed almost in sequence from the hilum of the thyroid into the upper pole of the thymus along the so-called stalk, or ligament, of the thymic lobe (*Fig. 295*). Indeed, these varicosities often extend into the substance of the thymus in the mediastinum (*Fig. 295*). The region of distribution of the lymph-cysts coincides with the region of distribution of thymic tissue from the hilum downwards into the mediastinum. That isolated, as well as compact, masses of lymph tissue in this region are thymic tissue is demonstrated by the finding, even in the

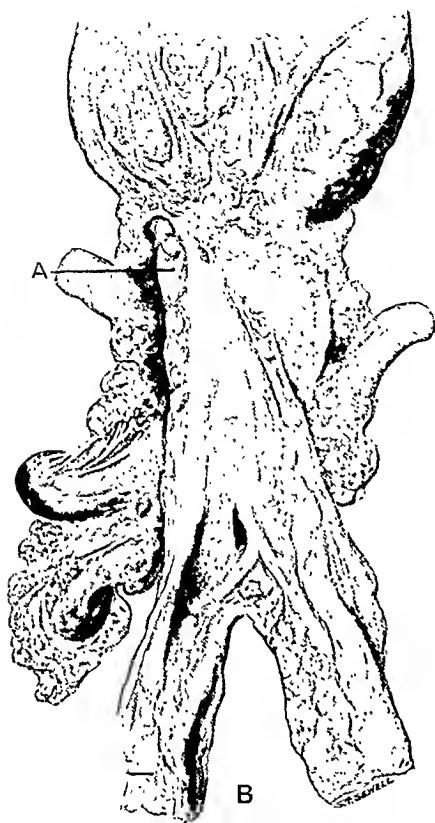


FIG. 295.—Drawing of thyroid and thymus showing the cord of lymphatics (Piersol's ligament) which extends between the two structures. Note the cystic distention on the course of the leash of lymphatics. Both leashes have undergone fibrosis, causing them to stand out as depicted here. A, Thyro-thymic ligament, showing a series of dilated lymph channels in its course; B, Lobes of thymus.

characteristics of lymphatic vessels (*Fig. 296*). Lymph is encountered in the channels. Frequently the component lymphatics, with care, can be dissected from the lateral surface of the trachea (*Figs. 297, 298*), upon which they lie, and traced to their origin in the hilum of the thyroid gland. The leash of lymph-vessels is always most prominent anteriorly, where it emerges from beneath the lower pole of the thyroid lobe (*Fig. 297*). On the other hand, these lymph-vessels may not be collected together into a cord or leash, but may spread



FIG. 296.—Drawing of dissection from foetus, made by Sir Astley Cooper, showing the stalk of the thymus, which he demonstrated to be absorbent vessels connected with the thyroid lobes. (Specimen 3730 D., R.C.S. Museum, London.)

hilum of the thyroid lobe, of characteristic Hassall corpuscles.

Careful dissection of the stalk of the thymus reveals the fact that it takes origin in the hilum of the thyroid lobe. This stalk, when it is well defined, is seen to be composed of vessels which have all the charac-



over the tracheal surface of the thyroid lobe and run down almost independently into the thymus. Thus it seems clear that there is a very definite lymph system which emerges at the hilum of the thyroid and passes directly into the lymph spaces of the thymic nodes. The sinusoidal lymph and migrating lymphocytes from the thyroid gland can at times be found in these lymph passages.

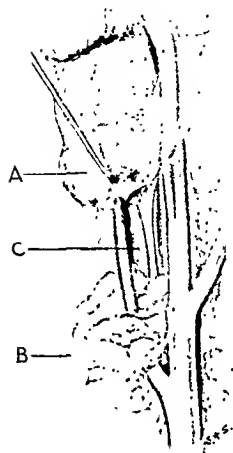


FIG. 297.—Drawing of leash of lymphatic vessels between the thyroid and thymus, dissected out to show its position on the anterolateral surface of the trachea, as it emerges from below the right thyroid lobe in its passage to the thymus. A, Right lobe of thyroid; B, Thymus; C, Leash of lymphatics.



FIG. 298.—Drawing of a dissection of the neck, showing thyroid and thymus *in situ*. The isthmus of the thyroid has been incised and the left lobe deflected from the trachea to expose the lymphatics passing from thyroid to thymus. A, Anterior leash of thyro-thymic lymph vessels; B, Upper pole of thymus; C, Body of thymus.

Confirmation of this special lymphatic system is obtained from 13 cases of cancer of the thyroid examined post mortem. In none of these cases were

the lymphatic glands of the neck, even those associated with the lower pole of the thyroid lobe, involved by the extension of the growth. In 9 of the 13 cases, however, a direct spread into the thymic tissue could be traced from the interstitial tissue lymphatics and from the invaded sinusoids (*Fig. 299*). This presence of a leash of lymphatics and thymic tissue in the fascia lying between the trachea and the thyroid has a most important bearing upon the direction of spread of cancer of the thyroid gland, and also upon certain other pathological conditions in which fibrosis may result in adhesions between the thyroid and the trachea upon which normally it moves freely.

It is generally accepted that, apart from these deep lymphatics, there are concomittant lymphatic vessels emerging with the veins on the surface of the gland. We have been unable to trace this system ourselves by the means at our disposal, but we do not doubt its existence.

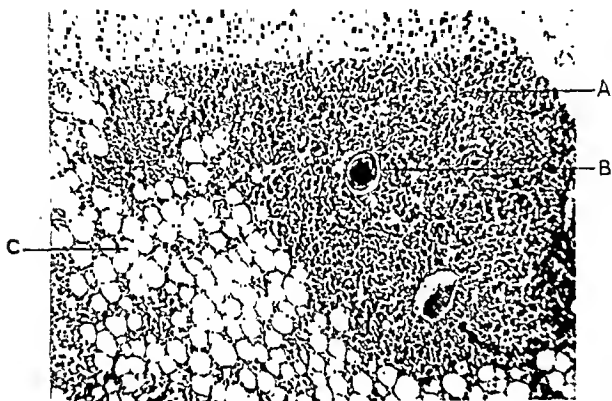


FIG. 299.—Showing the thymus invaded by cancer cells. From a case of carcinoma of the thyroid gland. A, Malignant cells; B, Hassall corpuscle; C, Fat tissue. ( $\times 100$ .)

We are concerned only to point out that anatomical, physiological, and pathological evidence is available to show that *the thyroid and the thymus are directly and intimately connected through the medium of a system of lymph-vessels, which emerge from the region of the hilum of the thyroid and enter the thymus at its upper pole.*

Sir Astley Cooper,<sup>10</sup> speaking of the cervical pole of the thymus, says, "I have generally seen it joined by vessels to the thyroid gland". Elsewhere he defines the arterio-venous connection, and makes it clear that these uniting vessels are 'absorbent' or lymph vessels continuous with the central reservoirs or ducts of the thymus, and contain 'chyle'. In the calf he succeeded in some measure in injecting the 'absorbent' vessels, and to-day some of his specimens are to be seen in the Museum of the Royal College of Surgeons.\* We are thus presenting a confirmation of work published as long ago as 1832 by a distinguished Fellow of this College.

#### THE THYMUS.

*Microscopic Anatomy.*—The thymus is a gland composed of endothelium. The endothelium is arranged in lobes or nodules, the individual cells of which also form the walls of a complex aggregate of venous and lymphatic capillaries. These lobules are held together extremely loosely in a definite capsule, very few strands of which penetrate into the substance of the

\* Nos. 3730x, 3730xA, Museum, Royal College of Surgeons, England.

gland. In man, in these strands of interstitial tissue are to be found the typical Hassall corpuscles. The thymic gland as a whole has a very poor arterial blood-supply. Such is the basic structure of the thymic tissue.

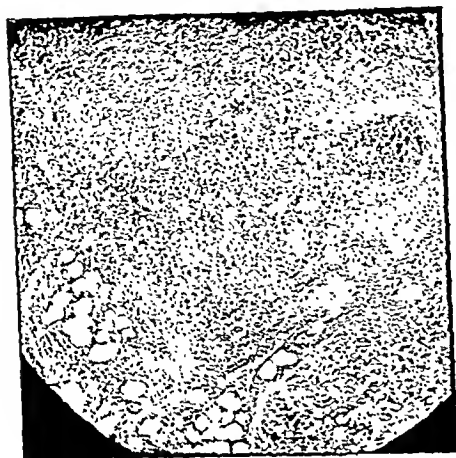


FIG. 300.—Section through the body of the thymus. The mixture of vesiculated fat tissue and lymphocytes represents two phases in the physiology of the gland. ( $\times 80$ .)

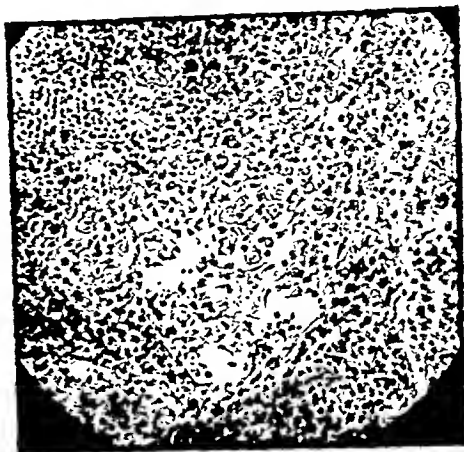


FIG. 301.—Showing the endothelioid or granular fat-phase in the human thymus gland. Note the large granular endothelioid cells and the relative diminution in lymphocytes in the surrounding tissue. ( $\times 170$ .)

The thymus is modified as regards its appearance by the physiological demands made upon it. This liability to variation, or metatrophy, is characteristic of the thymus and certain other endothelial glands. The morphological picture of the metatrophy of the thymic gland exhibits three varieties: (1) A lymphocytic variation (Fig. 300); (2) A granular or lipoidal fat variation (Fig. 301); (3) A vesiculated fat variation (Fig. 302). These pictures of the physiological changes are similar to those described by Gulland<sup>11</sup> and later by Job<sup>12</sup> in the so-called secondary lymphatic glands. They describe the conversion of vesiculated fat tissue into tissue bearing lymphocytes, and even going on to form the endothelial germ centres under the influence of activity in the adjacent (preformed) lymph glands. It will be seen therefore that the thymic gland is not exceptional in manifesting great differences in appearance in the course of its activity.

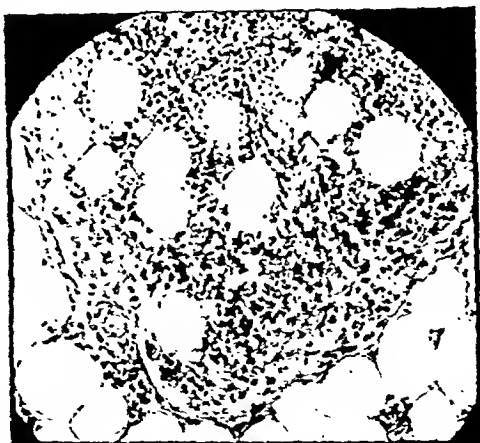


FIG. 302.—Sections through another part of the thymus showing the fat phase. Endothelioid cells and lymphocytes are relatively few in number, their place being taken by vesiculated fat. ( $\times 185$ .)

The vesiculated fat phase is the one commonly found in the adult. This fact explains the common view that the thymus suffers atrophy in early life. Apart from this, however, Hammar<sup>13</sup> has amply demonstrated that the typical lymphoid phase can be found at all ages in the human thymus.

The thymus may thus be described as an endothelial gland peculiarly associated with the venous and the lymph-vascular system. From its dynamic features it is definable as a lymph fat gland.\* We thus present a confirmation of the work of Kolliker, Toldt, Pende, and A. Kohn.<sup>14</sup> We have shown elsewhere that the lymphocytic activity occurs when the thyroid gland is most active,<sup>2</sup> and we look upon the thymus as a tissue which modifies the secretion, and stores any excess of secretion, of the thyroid gland.†

### PARATHYROID BODIES.

The identification of parathyroid bodies is always a matter of difficulty. They are most frequently confused with nodes of thymic tissue (*see Figs.*

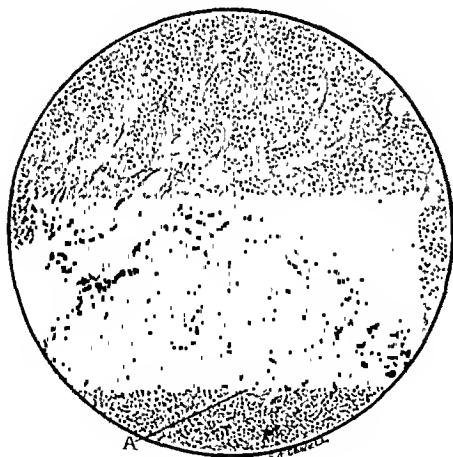


FIG. 303.—Drawing of a section of human parathyroid fixed and stained by the author's technique. The parathyroid body is shown together with the lymph tissue which by the presence of Hassall's corpuscles (A) can be recognized as thymic tissue. This was taken from the region of the hilum of the thyroid gland.

309, 310). The Hassall corpuscles, when present, serve to identify thymic tissue. We have found them present in nodes of tissue selected as representative parathyroid bodies. Except for the presence of the Hassall corpuscles, it is often difficult to distinguish between the endothelioid phase of thymic tissues and parathyroid tissue.

Parathyroid tissue is, in our experience, only typical when its tubular structure is apparent, when the tissue is compact, and has a definite arterial blood-supply. Apparently the parathyroid tissue can be scattered widely as microscopic fragments in the hilum of each thyroid lobe. When a parathyroid body is compact, the best guide to it is the parathyroid artery, which is a considerable vessel (Halsted<sup>16</sup>). Thymic nodes, on the other hand, have a trivial arterial supply. It is

also to be remembered that the parathyroid tissue is often embedded in masses of thymic tissue, so that almost any well-formed nodule in the hilum of the thyroid lobe may prove to be parathyroid (*Fig. 303*).

As a compact body the parathyroid seems to be the site of termination of a direct branch of the sympathetic nervous system (*Fig. 304*). Usually

\* It is perhaps appropriate to refer here to the recent work of Cramer<sup>15</sup> and others on certain 'lipoidal fat glands', in that they present metatrophic features comparable with those found in the thymus.

† An alternative to this is that the thymus is the storehouse of 'raw material' for thyroid activity.

the branch is derived directly from the superior or middle ganglion, although it may reach the parathyroid body through the medium of the very complicated plexus situated in the fascia on the posterior surface of the thyroid lobe (Figs. 309, 310). However the branch arises, it is frequently ganglionated. The ganglion may be on the nerve as it enters the parathyroid body with the artery (Fig. 305), or it may be embedded in the capsule of the parathyroid body. The parathyroid body, therefore, like the suprarenal and pituitary bodies, seems to be closely associated with the sympathetic nervous system, and deserves special investigation from this point of view.

The parathyroid body has a second feature worthy of note. This is the essential tubular arrangement of the parenchyma. Under normal conditions evidence of this is found in an occasional tube containing the fluid, lymph-like material (Figs. 306, 307, 308). When this appearance is prevalent in the parathyroid tissue, it is definitely associated with secretory activity in the thyroid tissue, when the



FIG. 305. - Human parathyroid artery (B), alongside which is the ganglionated branch of the sympathetic (A) going to the parathyroid body. ( $\times 50$ .)

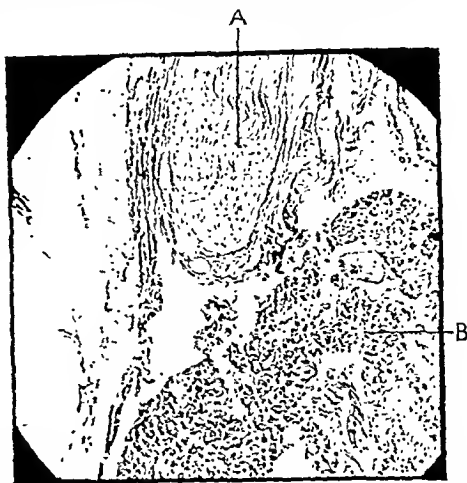


FIG. 304. - Showing a branch of the cervical sympathetic lying alongside the parathyroid body prior to the distribution of its filaments in the substance of that organ. A, Sympathetic; B, Parathyroid. ( $\times 75$ .)

tissue is stained by the author's technique.<sup>2</sup> The same type of fluid, and occasionally lymphocytes, occupy both the lymph spaces of the thyroid gland and the tubes of the parathyroid body. In extreme degrees of parathyroid activity it would seem possible to trace the continuity of this fluid substance from the thyroid to the parathyroid. This fluid in the parathyroid body has in the past been taken to imply the identity of the thyroid and parathyroid epithelium. This we have already demonstrated not to be the case.<sup>1</sup> Further, in the literature it is generally conceded that the content of the parathyroid tube is not true colloid, but a thin granular

fluid lymph. This we identify with *secretion* in the lymph spaces of the thyroid. Moreover, the parathyroid in man seems to be constantly associated with one or more thymic nodes which participate in changes occurring in

the parathyroid. Extreme degrees of hypertrophic activity of the thyroid tissue (so-called hyperplasia) may coincide with the fairly complete conver-

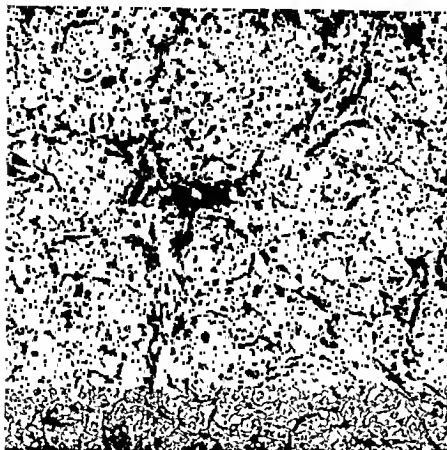


FIG. 306.—Illustrating the general structure of the human parathyroid, showing the endothelial stroma enclosing the tubulate lobules of the parenchyma. ( $\times 150$ )

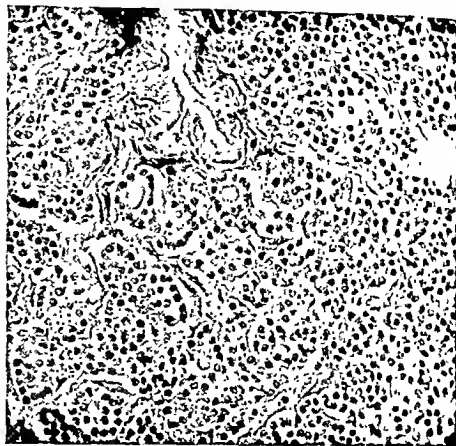


FIG. 307.—Human parathyroid gland, showing tubular nature of its structure. Two tubes are shown containing material similar to the secretion produced in the thyroid during secretory activity. ( $\times 180$ )

sion of the parathyroid body into a mass of follicular tissue (Fig. 308). This occurs in the embryo with great frequency, and in the adult occasionally, and it is a mistake to look upon these conditions as pathological.

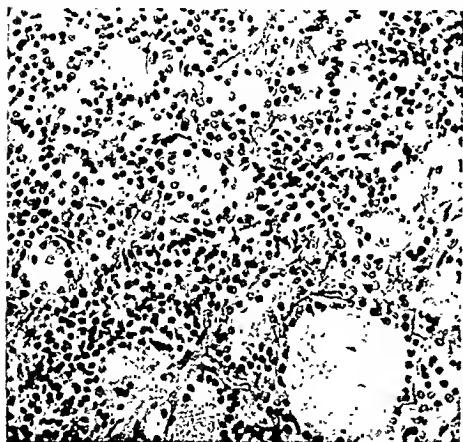


FIG. 308.—The same parathyroid body as in Fig. 307, stained by author's technique. The appearance of follicles and the refractility of their contained material suggest thyroid tissue, but the epithelial columns contain no central microcapillary system shown in all thyroid tissue stained with this technique. ( $\times 185$ )

It will be seen therefore that either the parathyroid tissue concurrently with the thyroid tissue accumulates secretion, or that the parathyroid body receives from the thyroid gland some of its secretion en route to the special thymic nodes to which the parathyroid is attached. We adopt the latter point of view, because we have not been able to find fluid accumulations in the parathyroid in the absence of secretory activity in the thyroid gland.

Further, lymphocytes are produced in the thyroid gland during secretory activity, and can be traced, via the perivascular lymphatics of the gland, into the tubular structures of the parathyroid body.

The conclusion we reach is that the parathyroid and thyroid glands work hand-in-glove in the execution of some common purpose.

SUMMARY OF ANATOMICAL CONSIDERATIONS (*Figs. 309, 310*).

1. A lymph sinusoid is described as the basic architectural feature of the thyroid gland. Its physiological morphology is explained.

2. The conception receives support from studies in embryology and comparative anatomy.

3. The functional arterial supply of the thyroid enters the gland at the hilum: it is derived from the inferior thyroid artery.

4. A subsidiary arterial supply (possibly nutrient) is derived from the superior thyroid artery, which usually contributes but little blood to the functional gland-units.

5. A 'shunt circuit' vascular mechanism is shown to exist in the thyroid gland.

6. A thyro-thymic leash of lymphatic vessels is demonstrated. This is shown to arise in the hilum of the thyroid and to pass down, on the anterolateral surface of the trachea, into the thymic lobes.

7. Lymph drainage of the thyroid is discussed, and it is suggested that the thymus gland is a reservoir for thyroid products.

8. The thymus is shown not to undergo atrophy in youth, but to persist and manifest a metamorphism co-incident with the nature of the demands made upon its function. It is analogous to other lymph fat-glands.

9. The parathyroid body is associated with a ganglionated branch of the cervical sympathetic nerve.

10. The parathyroid body is tubular in arrangement; at times the lumen contains material similar to the lymphlike secretion of the thyroid.

11. The probability is discussed of this material having been passed on to the parathyroid body from an actively secreting thyroid gland.

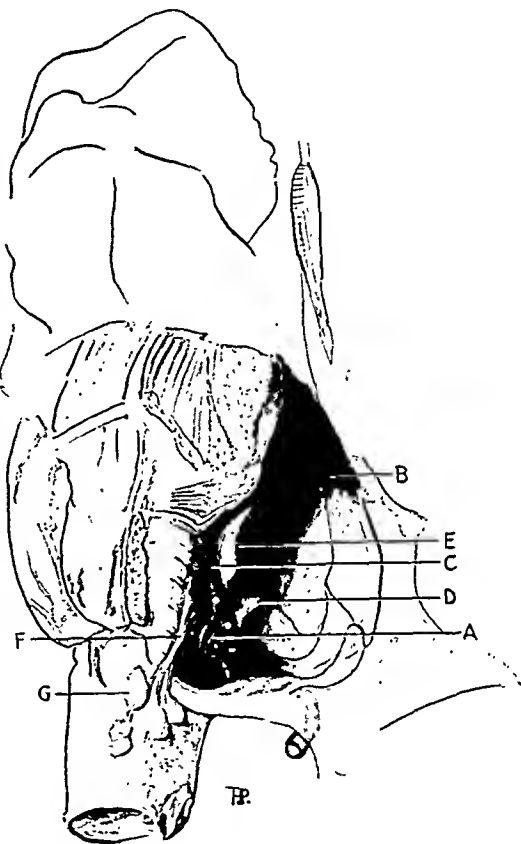


FIG. 309.—Drawing of the fascial bed of the left lobe of the thyroid gland to show the relative positions of the structures occupying the hilum of the thyroid lobe. A, Inferior thyroid artery; B, Branch of the sympathetic nerve; C, Recurrent laryngeal nerve; D, Parathyroid body; E, Lymph-fat gland body (thymic tissue); F, Thyro-thymic leash of lymphatic vessels; G, Thymic tissue.

## PHYSIOLOGY.

In our earlier studies of the thyroid apparatus<sup>1</sup> we have shown that there are two processes that occur in the normal thyroid gland. One is the passive accumulation of colloid, or colloid storage, the other is the active process

of secretion. These are distinct functions, and colloid is a substance different from secretion.

Hürthle,<sup>3</sup> writing in 1894, recognized that there were two different processes in the epithelium, each one leading to the accumulation of material within the follicle.\* Hitherto his original suggestion that these appearances

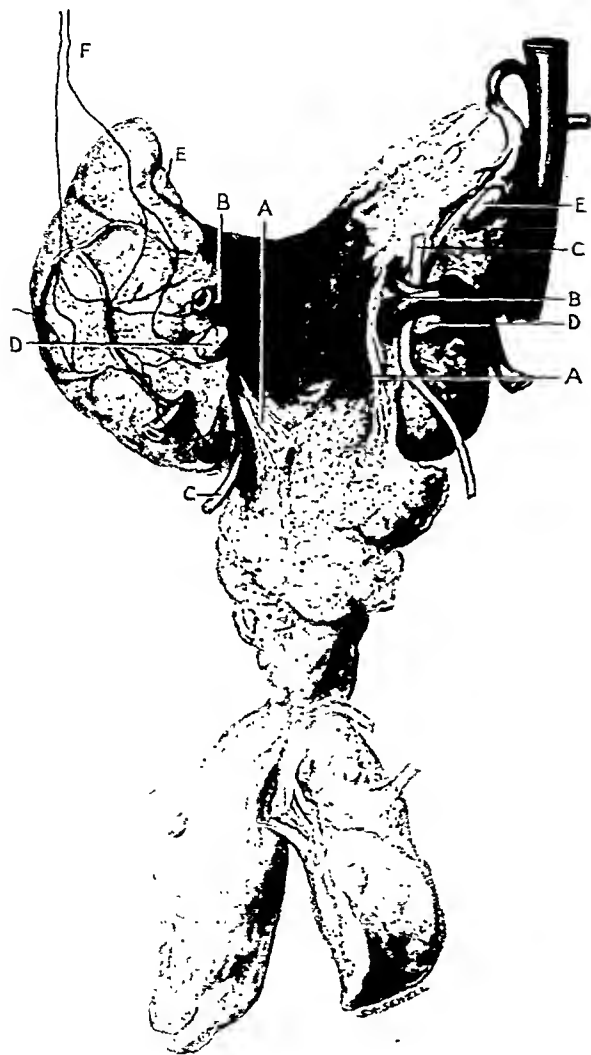


FIG. 310.—Posterior view of the thyroid apparatus, showing the relations and connections of thyroid, parathyroid, and thymus glands. Drawn from a dissection. A, The emerging plexus of lymphatics uniting thyroid and thymus; B, The inferior thyroid artery breaking up into its terminal branches in the hilum of the gland: note the cut œsophageal branch and the trivial anastomotic contribution from the superior thyroid artery; C The inferior laryngeal nerve, showing its relation to the posterior border of the hilum of the lobe, the inferior artery and its branches, and the parathyroid body; D, Parathyroid body; E, Thymic nodes; F, Sympathetic nerves forming a plexus in the fascia over the posterior surface of the lobe, contributing twigs to the gland via the inferior artery, and a ganglionated branch to the parathyroid body.

could not be reconciled as the expression of one function has perforce been neglected, and the production of colloid has always been regarded as the end-result of all activity. As long as the simple follicle was accepted as the

\* "Bei der Secretbildung sind nach meinen Beobachtungen zwei verschiedene Formen der Secretion zu unterscheiden, nämlich (1) Secretbildung des Follikelepithels mit Erhaltung der Zellen und (2) Secretbildung durch Untergang von Zellen".



unit of function, this was inevitable. When, however, the larger area of the gland-unit was recognized as the unit of function, it at once became obvious that colloid and secretion were different substances, and that colloid was not the result of activity in the tissue in their so-called 'hypertrophic' state.

**The Process of Colloid Storage.**—We have shown that the accumulation of colloid within the epithelial column can be followed from the first microscopic droplet to the distended vesicle of the normal gland in the colloid-storage phase of its activity. In every stage of colloid formation examined histologically, the epithelium, the sinusoid, and the endothelium of the gland-unit all maintained a perfectly uniform and constant appearance of quiescence. Furthermore, in a gland-unit in which colloid storage is in process there is no evidence of production of secretion. It would seem necessary for the epithelium to evacuate the stored colloid and return to the solid form prior to the production of secretion in that same gland-unit. This sequence of events was first observed in the experimental researches of McCarrison before being put forward by us.

**The Process of Secretion.**—In this process every element of the gland-unit takes active part. The epithelial nuclei, the cytoplasm, and the micro-capillary system all become laden with granules. This granular material ultimately liquefies, and occupies the centre of the epithelium, thus forming an accumulation or 'lake' of secretion in the column. At the same time the endothelial cells of the sinusoidal capillaries become laden with granules, and a thin fluid and lymphocytes appear in the sinusoid itself and in the perivascular lymphatics surrounding it.

The picture of a gland-unit in secretion is thus a totally different one from that of a gland-unit storing colloid. Next to a gland-unit which is in process of secretion there may be other gland-units in the process of storing colloid.<sup>17</sup> One gland-unit, however, is only occupied in carrying out one function at once. It may be argued that, although the two processes do not occur together in the same gland-unit, nevertheless the secretion formed in one may pass on to an adjacent gland-unit for storage in an altered form as colloid. We have not discovered in the gland any anatomical channels whereby such transference could be affected. It certainly does not occur through the medium of the lymphatic channels. The lymph channels and sinusoids never contain colloid during the process of colloid storage, while the fluid within the sinusoidal spaces during secretion has a similarity to secretion itself.

**Significance of the Two Functional Cycles.**—Colloid is not secretion. We have already set forth elsewhere<sup>18</sup> reasons for the probability of colloid being of the nature of a carrier or vehicle which, like the taurocholates and glycocholates of the bile, is continually in circulation in the body. Only the excess or reserve of bile is stored in the gall-bladder. We suggest that only the reserve of colloid is stored in the thyroid gland by the process of colloid storage.

We have already described elsewhere<sup>15</sup> the decades in the life of the individual at which secretion is the main, though never the sole, activity of the thyroid mechanism. In the embryo and the new-born, secretion seems to be the only product of the gland. It is well to remember that at this

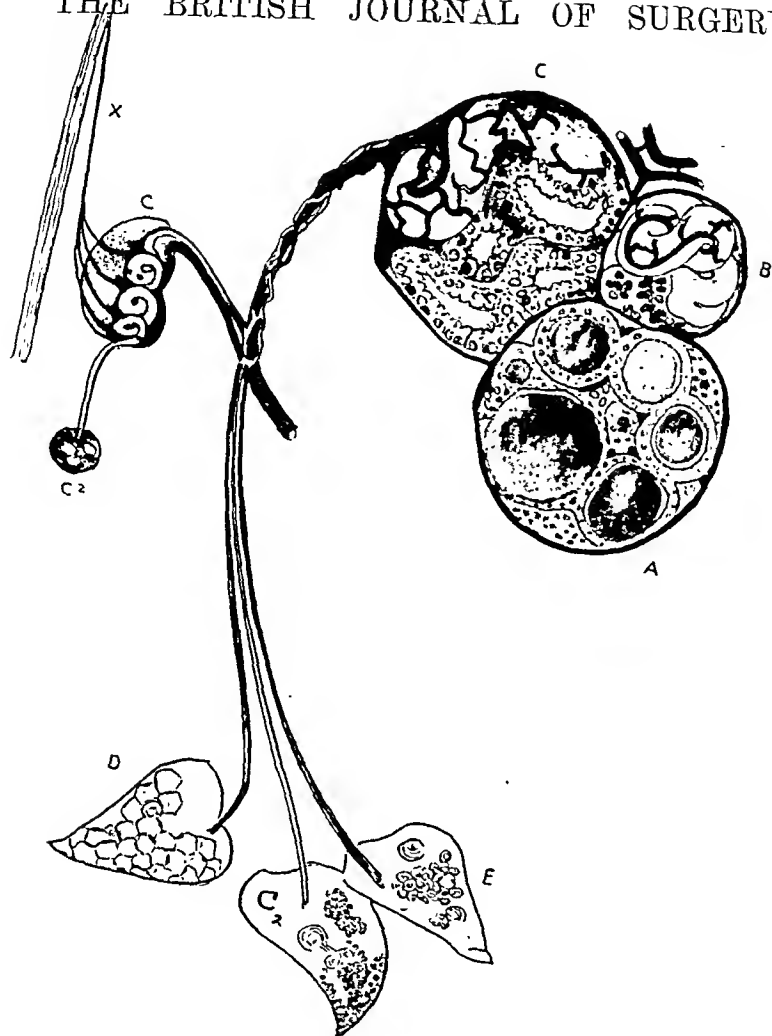


FIG. 311.—Diagrammatic summary of the histophysiology of the thyroid apparatus. A, A gland-unit engaged in accumulating and storing colloid by the process of vesiculation. This occurs in defined areas of the epithelial columns, and leads to fragmentation of the columns into tense discrete spheroidal vesicles. The ground tissue of the gland-unit appears as a pavement of epithelial cells, representing the roofs or floors of vesicles in tangential section. No demonstrable biochemical disturbance of the nuclei or protoplasm of the epithelium accompanies vesiculation. Such a gland-unit must arise from, or revert to B, the rest phase of the thyroid unit. This phase has long been recognized as the loosely termed 'fetal rest tissue'. It does resemble embryonic thyroid tissue, but it is the common state of the thyroid in starvation and marasmus, and may occupy the whole gland in some normal individuals whose turnover is at a low level. The nuclei are orientated in an indefinite manner about the central intra-epithelial microcapillaries, in contrast to the regular pavement arrangement in the colloid unit. This rest phase always intervenes between the colloid stage and C, the secretory phase. The sequence of events in the transition from rest to activity seems to be as follows. The sinusoid opens and contains a fluid and lymphocytes; the nuclei move to the sinusoidal surface of the epithelial column; the protoplasm becomes granular and later vacuolated; the vacuolar fluids flow to the central zone, forming a lake (the process of lacunation); there is no tension in the cavity so formed. The microcapillaries fill with fluid and granules. Secretion so formed is not stored in the follicles, but appears in the lymphatics leading from the sinusoid; in the parathyroid (C<sup>1</sup>), where it gains intimate contact with the sympathetic nervous system X; and in the lymphatics leading to the thymus, in which it induces a lymphoid phase. The thymus can be traced through three phases: a vesiculated fat D, a granular lipid E, and a lymphocytic activity (C<sup>2</sup>). It is only in the secretory phase that the co-operation of thyroid, parathyroid, and thymus is evident.

age the gland is devoid of iodine. After birth colloid storage increases up to five years of age, while secretion declines. From nine to fifteen years the position is reversed; secretion predominates in the work of the gland, and may overshadow the colloid-storage function. Thereafter up to eighteen to twenty years, sex determines the picture; the female presents in the main a secretory picture, and the male in the main one of colloid storage. After that age the female approximates to the male type, except in pregnancy and perhaps in menstruation. In his recent book, de Quervain, speaking of secretory activity as 'hyperplasia', seems to confirm this distribution of the alternation of the two processes in the gland at different ages.

The above summarizes the condition in the average individual. It must be remembered, however, that this average covers very widely separated extremes. Every individual seems to be a law to himself in the demands made upon the secretory activity of his thyroid apparatus; a physiological enlargement may be a necessity. We would remind our readers that the liver, the kidneys, and the lungs—in fact, all the organs—conform to this last rule. It is perhaps fortunate that swellings in these organs are less obvious than those of the thyroid gland, and that it is thus less possible for the uninitiated to measure their varying morphology in pathological terms.

Before passing on to the clinical application of our work, it is necessary to emphasize the fact that any excess of secretion of the thyroid gland is stored mainly outside the thyroid, in the thymus, and it may be in all lymph-endothelial tissue. Normally, at the height of the secreting process, slight accumulations of secretion are found in the centre of the epithelial columns (lacunation of secretion). Any delay in the evacuation of this secretion from the follicles is unusual in the normal gland. Indeed, the whole pathology of secretory activity seems to turn upon the failure of the tissues to effect a natural re-absorption of the secretion. We would point out that, according to modern views, a similar failure on the part of kidney parenchyma to re-absorb is the basic factor in the pathology of nephritis.

For the clinician the important points to remember are (*Fig. 311*): (1) That secretion is not colloid; (2) That intra-glandular storage of secretion is rare in the normal gland; (3) That colloid is a separate entity; (4) That there is an essential need to maintain in the gland a reserve of colloid which on demand is free to circulate in the body generally.

It is possible now for clinical purposes to trace, in the exaggerations and disorders of these functions, the significance of certain common forms of goitre.

### APPLIED PATHOLOGY.\*

A detailed study of the pure pathology of the goitrous thyroid gland has already been published by us<sup>17</sup>, and should be consulted for a fuller understanding of the morbid physiology and anatomy underlying the various goitres. It is characteristic of the 'visceropathies'<sup>18</sup> in general that the associated symptom-complex is an expression of the failure of the

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\* The following sections form part of a thesis presented for the M.D. Edinburgh, and awarded a gold medal.

compensatory mechanism. One symptom-complex may thus follow a variety of pathological conditions (e.g., in renal and cardiac disease). This also appears to be the case in the diseases of the thyroid gland. Because of this, it is seldom possible to present a satisfactory classification of the clinical conditions if the clinical nomenclature of diagnosis is a mixture of 'pure' and 'applied' pathology. When a simple constant morbid state underlies a constant clinical syndrome, pure and applied pathology may coincide; but even under these conditions it is not safe to assume that the pathological condition in the organ is the etiological factor in the production of the symptoms—i.e., that one is the cause and the other effect. This is, of course, well recognized in the diseases associated with renal disturbances, but is less well recognized with the so-called endocrine glands. The fact that insulin exercises a profound influence in diabetes has not established the pancreas as the primary and only seat of the disease of which diabetes is the symptom. Any of the many 'thresholds', as the physiologists term them, may be the seat of the damage—the threshold of 'absorption', of 'co-ordination', of 'utilization', or of 'excretion'. We are as yet in ignorance of the body function over which the thyroid apparatus presides, so that we know nothing of various 'thresholds' of activity and exchange concerned. Without this knowledge we cannot hope to reach a satisfactory understanding of the etiology of the diseases associated with goitre. Indeed, we cannot even approach the problem until we recognize the foregoing fact, and cease to be content with the vague satisfaction provided by the 'endocrine' theories.

#### DISORDERS OF SECRETION.

Two disorders of secretion demand particular consideration: (1) *Graves' disease*; (2) *Simple thyrotoxicosis*.

**1. Primary Graves' Disease.**—Graves' disease is associated with many pathological types of goitre. Berry,<sup>20</sup> Plummer, Wilson, and Boothby<sup>22</sup> have already shown that Graves' disease proper has a distinctive pathology.

We have recently demonstrated that there is one type of goitre in which this specific histopathology occupies the *whole* of the substance of the thyroid gland. Such a goitre we have termed 'adenoid goitre'.<sup>17</sup> Clinically, adenoid goitre has a 100 per cent coincidence with the full syndrome of Graves' disease. Pathologically, adenoid goitre has a second 100 per cent coincidence with the *absence of colloid vesicles* in the gland.

If we look at the histology of adenoid goitre, three distinct and easily recognizable stages appear in its progress. In the first stage the tissue is uniformly in a state of secretory activity, while the physiological factor of vesiculated colloid (i.e., colloid storage) is absent. According to our studies of the normal physiology of secretion, it is essential for the balanced action of the gland that colloid should circulate through the parenchyma. It seems natural to conclude that the absence of any reserve of colloid would inhibit the proper production of secretion: thus secretion produced in the absence of colloid cannot exercise its normal effect.\* The action of such a secretion

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\* This defect, as Boothby suggests, may be due to a lack of available iodine with which we know the colloid to be associated.

upon the body is unphysiological. The disturbance from a histopathological point of view is a secretory dysthyroidism—a conclusion which has already been reached by many observers on purely clinical grounds.

There is, moreover, another point of importance in the light of our conception of the thyro-thymic mechanism. Post-mortem examination of cases of adenoid goitre reveals the fact that, although the thymus is enlarged, at no stage does it contain an excess of lymphocytes. It is, on the contrary, in the endothelioid phase of its physiology. The thymic reservoir presumably, as indicated by the absence of the lymphocytes, is not being called upon to store any excess of the secretion that is so obviously being produced in the thyroid gland. This absence of lymphoid hypertrophy in the thymus has also been commented upon in particular by Gebele,<sup>23</sup> Halsted,<sup>24</sup> Crotti,<sup>25</sup> and Falta,<sup>26</sup> and is still further emphasized by many Continental observers, who separate from true Graves' disease a 'thymic Basedowii' which has its own peculiar pathology. The significance of this abnormal participation of the enlarged thymus in the inordinate activity of the thyroid in Graves' disease with an adenoid goitre would seem to point to the probability that all the viciously produced secretion is being utilized as it is produced. In Graves' disease associated with adenoid goitre histopathology indicates that both demand and supply are exalted to a pathological degree. This would imply that the cause of adenoid goitre lies in some factor extrinsic to the thyroid gland.

The next stage in the progress of adenoid goitre is the filling up of the follicles until, under the microscope, they are seen to be choked with secretion. When this occurs the sinusoids become empty, and few if any lymphocytes are found in the gland. This choking of the follicles probably means the forcing of secretion through unusual channels: for example, the blood-channels. It is perhaps analogous to the jaundice which follows choking of the bile-passages in the liver, or to the uræmic state which follows the choking of the kidney in parenchymatous nephritis. Clinically this second stage is signalized by the exacerbations of acute symptoms which occur in the course of the disease, and which may decline with treatment by rest. That is to say, the exacerbations represent, according to our view, the addition to the initial exophthalmic syndrome of a thyrotoxicosis, which is the result of the regurgitation of secretion from choked follicles, perhaps into the blood instead of into the lymph.

The third stage in adenoid goitre begins when a diffuse fibrosis of the walls of the sinusoids circumscribes the follicles as an impermeable membrane. The blood capillaries, however, lie between the fibrous wall and the 'naked' epithelium. This renders evacuation of secretion impossible through normal channels; the choking of the gland-unit becomes irreversible. The thyrotoxicosis then becomes continuous, and clinically the disease loses its exacerbative features. This third stage is the one in which partial extirpation of the gland is found to be satisfactory. The explanation is that the operation removes a mass of gland substance which contains, pent up within its fibrotic follicles, the perverted secretion which is toxic in its action. In this stage surgery is performing more quickly what nature is slowly accomplishing by atrophy and exhaustion of the epithelium.

Thus the pathology of adenoid goitre contains for the clinician the suggestion that primary Graves' disease presents the following sequence: (1) An 'exophthalmic' stage; (2) A superadded exacerbative thyrotoxic stage; (3) A more or less continuous thyrotoxic stage proceeding to atrophy and exhaustion. Each of these stages has its specific histology.

Such a sequence of events associated with a typical adenoid goitre we would term primary Graves' disease—primary, in that the cause is extrinsic to the gland.

**2. Simple Thyrotoxicosis.\***—There is a thyrotoxicosis uncomplicated by exophthalmos. We think the term thyrotoxicosis (Koehler)<sup>27</sup> is preferable to such terms as 'forme fruste', 'Graves' disease without exophthalmos', 'toxic adenoma', etc., inasmuch as there is a specific pathology underlying the condition wherever it occurs. The condition may follow any of the many pathological changes which give rise to goitre. It has already been discussed as a specific part of the pathology and clinical syndrome of primary Graves' disease.

In the thyroid gland which is the seat of a progressive fine sclerosis, atrophy and degeneration of the gland substance take place. In a varying percentage of these cases this loss of gland substance is countered by a compensatory hypertrophy of the healthy *residuum* of the gland. It is this residuum of functioning tissue that provides the pathological basis for the thyrotoxicosis, and not the primary lesion, which is merely destructive. The compensatory hypertrophy may be due to excess of supply of metabolites from which secretion is produced, or to an excess demand for secretion; neither is absolute; both are relative to the available healthy gland tissue.

If attention is focused upon this residuum of gland substance, it will be seen that the continual progress of secretory activity may ultimately lead to the choking of the follicles with secretion; that is to say, by distention of the follicles the sinusoidal space is obliterated. On the assumption that was made for primary Graves' disease, we look to the regurgitation of this accumulated secretion as the cause of the thyrotoxic symptoms. The fundamental histopathology of simple thyrotoxicosis is thus the same as that found in the second stage of primary Graves' disease, viz., the accumulation of secretion distending and choking the follicles. In primary Graves' disease, however, the condition is general throughout the gland, whereas in simple thyrotoxicosis it is a focal one.

There is a difference as well as a similarity between these two conditions, for it must be noted that in simple thyrotoxicosis *colloid does not disappear* from the gland as a whole. It appears therefore that the overworking of the residuum of the tissue can lead to a choking of the follicles with secretion, and to a vicious resorption of the contents even in the presence of a sufficiency of colloid. Clinically the condition is associated with intoxication, and generally with goitre. The condition, more often than not, stops short at the thyrotoxicosis, constituting the so-called 'toxic adenomatosis' of Plummer<sup>28</sup> or the 'interfollicular adenomatosis' of Goeteh.<sup>29</sup>

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\*The word toxic is used here in a general rather than in the specific sense of the bacteriologist and pharmacologist.

Obstruction to the normal outflow of any excess of secretion is the fundamental pathology of simple thyrotoxicosis. On this basis a mechanical obstruction may occasion this result, and it may be that a simple tumefaction, by its bulk or its disposition within the gland, can result in simple thyrotoxicosis—toxic adenoma, or, as it would more accurately be described, adenoma complicated by thyrotoxicosis.

**Secondary Graves' Disease.**—Secondary Graves' disease is an immediate or remote sequel of a simple thyrotoxicosis.

The basic pathology of secondary Graves' disease is the same as that of simple thyrotoxicosis, in that it is the result of a compensatory hypertrophy in the functioning residuum of a gland which is the seat of some diffuse destructive lesion. Thus, like simple thyrotoxicosis, it represents a condition of affairs in which the effective or normal production of secretion is interrupted. In the course of the progressive condition, the hypertrophic residuum of tissue itself suffers from a fibrosis. This fibrosis is essentially of a different order from the primary lesion in the gland. It is the result of the overworking of the functioning residuum of thyroid tissue, and is of the order of 'strain fibrosis' described by Adami.<sup>19</sup> It is of the intimate variety which penetrates into the gland-unit and insinuates its fibres between the epithelial and endothelial surfaces of that unit. This results in the production of a fibrotic wall about the follicles. The strain of overwork has been operating on the whole residuum; colloid-storage areas and secreting areas alike are obstructed, so that colloid also becomes pent up within the fibrotic vesicles of the residuum of tissue. Colloid, therefore, though obviously present in the gland, *is now rendered unavailable for the purpose of function and circulation.* We return thus, but by a different route, to the condition of affairs that we have demonstrated in primary Graves' disease—namely, the presence of pent-up secretion within the epithelial columns, and the absence of circulating colloid. The difference between the primary and secondary Graves' diseases is that in one the histopathological condition in the gland is general, while in the other it is focal.

Naturally in all cases clear lines of demarcation between the individual events in the sequence which leads to secondary Graves' disease cannot be seen. For example, in secondary Graves' disease of the young, an early 'puberty goitre' can induce a perifollicular fibrosis. We have seen that the physiological demand for secretion in the female at this age is considerable. The residuum of the gland may therefore be compelled to undergo an extremely rapid hypertrophy, so that the preliminary thyrotoxicosis is masked by the rapidity of the progress of the disease. As a contrast to this, the secondary Graves' disease of the senile period (e.g., menopause) usually has years of simple thyrotoxicosis before exophthalmos ensues.

In a well-marked case of secondary Graves' disease this sequence of events is in marked contrast to that of primary Graves' disease, in which the exophthalmos occurs conspicuously early, and toxic symptoms follow later.

Pathology suggests that the main features of secondary Graves' disease are: (1) A preliminary exacerbative thyrotoxicosis; (2) A chronic, or

continuous thyrotoxicosis; (3) The supervention, at a later date, of exophthalmos to complete the syndrome. This is the inverse sequence to that of primary Graves' disease.

**Discussion.**—The pathology of function of the thyroid gland is identical in Graves' disease, whether this be of the primary or secondary variety, and wherever it be found in goitrous conditions.<sup>17</sup>

If the morphological appearances of the thyroid tissue in the two diseases be examined, this identity is not apparent. In the gland of primary Graves' disease (adenoid goitre) no colloid can be seen in the tissue, while in secondary Graves' disease (intralobular fibrotic goitre) varying amounts of vesiculated colloid can always be found, but each vesicle is surrounded by a fibrotic wall which renders the colloid of no avail from the point of view of function. Thus in both types of Graves' disease the disorders of function in the gland are the same, and consist in a perversion of secretory activity together with an absence of colloid capable of circulation through the gland. In neither case is the disorder of the nature of a 'hyperthyroidism'.

From our observations of the pathology of the two conditions, we are driven to the conclusion that the essential difference between primary and secondary Graves' disease is to be found in a critical examination of the *relation of the demand made upon thyroid activity to the capacity for response in that tissue*. In primary Graves' disease we must postulate an initial inordinate demand made by the body generally upon a normal and willing thyroid gland, while in secondary Graves' disease the condition is one of a normal demand made upon a previously injured thyroid gland. From what direction the insatiable demand upon the thyroid in primary Graves' disease comes we are quite unable to state, and can but suggest that the demand arises in some metabolic disorder analogous to, and as intricate as, that which underlies the disorder of carbohydrate metabolism in diabetes. We would suggest that this possibility in itself should be sufficient to cause the physician to return into the field of thyroid disease, and renew his attack upon its early manifestations.

It is to be noted that the lesion in secondary Graves' disease does not preclude the possibility of a sufficiency of colloid somewhere in the body, though this has not yet been demonstrated. It may be that iodine metabolism is still possible outside the thyroid gland, as Boothby suggests.

With regard to the underlying cause in the thyroid gland which ultimately makes the onset of secondary Graves' disease possible, we are equally uninformed. The pathology of the condition is analogous to that of chronic interstitial nephritis. Thus, in this branch of thyroid disease also, it is to the clinician that we must look for an elucidation of the earlier factors in the etiology of the disease.

In these conclusions, based upon histopathology, lies a satisfactory substantiation of the work of the Mayo Clinic. Plummer, Wilson, and Boothby seemed to have reached a similar, if not identical, differentiation of the types of Graves' disease from the clinical and pharmacological point of view.

It will be seen that in all these conditions the basic pathology of Graves' disease and thyrotoxicosis implies a degree, it may be a considerable one,



of *hypothyroidism*<sup>17</sup> (not hyperthyroidism) in the presence of the *dysthyroidism*.<sup>30 31</sup> That is to say, there is an *insufficiency* as well as an *inefficiency* of the secretion.

*Goitrous Tachycardia*.—Thyrotoxicosis we have seen to be a factor common to both types of Graves' disease, and to be a clinical condition of itself. At this point it is as well to insist that the tachycardia that arises in many forms of chronic goitre (e.g., interstitial thyroiditis) is not necessarily the result of a thyrotoxication; nor is the anxiety and agitation secondary to the tachycardia at all related to the psychosis and tremor of true thyrotoxicosis. The workers of the Mayo Clinic emphasize this fact, and seek an explanation in some form of irritation of the sympathetic nervous system. If we recall the heavy sympathetic innervation of the thyroid gland, it seems not at all improbable that a simple interstitial fibrosis may irritate the nerves, or that retrogressive changes which occur in the gland substance may also involve the intimate nerve-fibres, inducing a form of neuritis, apart altogether from any thyrotoxicosis.

**Summary of Disorders of Secretion.**—Primary Graves' disease, secondary Graves' disease, and simple thyrotoxicosis have certain histopathological features in common, just as they have certain clinical features in common.

*Primary Graves' Disease*.—Primary Graves' disease affects the whole gland, producing an adenoid goitre. Some extrinsic cause for the change in the gland seems certain. The stimulus to secretion is overwhelming, absolute, and not relative. Colloid disappears from the gland, and later becomes insufficient to serve the body function. In the beginning the body completely utilizes the excess of viciously produced secretion. Later the body fails to utilize it; it becomes pent up in the follicles, and the excess overflows, to exercise a disturbing 'toxic' action on the tissues.

*Thyrotoxicosis*.—Thyrotoxicosis is always secondary to some gross primary lesion which reduces the functioning power of the gland. The stimulus to secretion is only relatively increased; loss of thyroid substance is the cause of this relative increase. Colloid in the glands, and perhaps also in the body, still remains sufficient to serve the body function. The amount of secretion becomes greater than the gland mechanism can eliminate. It is pent up in the follicles and overflows as a disturbing 'toxic' substance.

*Secondary Graves' Disease*.—Here also the stimulus to secretion is only relatively increased owing to the loss of gland substance due to previous injury. But in this case there is also an interference with the efficient circulation of colloid through the gland. The secretion is similarly shut up in the follicles, and again it overflows to exercise a disturbing 'toxic' effect.

#### DISORDERS INVOLVING COLLOID STORAGE.

**Acute Vesicular Goitre.**<sup>17</sup>—In studying the applied pathology of adenoid goitre, we noted the entire absence of colloid from the gland, and constant association with the symptoms of Graves' disease.

Acute vesicular goitre or colloid goitre has the inverse picture, an entire absence of secretory activity and the entire absence of subjective symptoms.\* In these facts alone there seems to us to be convincing proof of the duality of functions in the thyroid gland. To credit colloid with the properties of a secretion, as evinced in Graves' disease, and at the same time to term the huge acute swelling (vesicular goitre), in which we know there is a vast increase in the actual amount of colloid, a sign of hypothyroidism, seems to be a contradiction in facts as well as in terms. This peculiar pathological condition, acute colloid goitre, is associated with a peculiar negative syndrome. Berry<sup>32</sup> was the first to note that acute colloid goitre is associated with a high incidence in the young male. Since we have been able to exclude from the group of colloid goitres the minor degrees of hypertrophic goitre (the old adeno-parenchymatous goitre—see p. 493), we find the incidence of acute colloid goitre in the young male almost as striking as the incidence of primary Graves' disease in the young female. The surgeon is familiar with this comparatively rare acute puberty goitre, since it is usually presented to him as an emergency matter—acute dyspnoea—presumably arising from the impaction of the descending swelling in the bony ring of the thorax. (Chronic goitres, of course, can be responsible for a compression dyspnoea.) It has been objected that the female escapes operation, and in that way the disproportion of incidence between the sexes arises. This explanation seems to us quite untenable. There is no anatomical reason in the female why an acute thyroid swelling should not descend and cause acute compression and dyspnoea. Indeed, from the high incidence of all goitres in the female, we would anticipate the reverse, unless, as we suggest, the acute colloid goitre is of greater general frequency in the young male at puberty as a specific condition. There can be no doubt that in this colloid enlargement, in its acute forms, the colloid material is pouring into the follicles of the gland. If colloid were produced in the manner of any secretion, the epithelium must manifest the extraordinary changes in form and content associated in other glands with the elaboration of secretion. Yet nothing looks so placid and passive as the epithelium; likewise, the sinusoids are empty, the lymph channels are not in evidence, and the endothelium of the distended capillaries is in a quiescent state. Everything, in fact, makes it clear that we are dealing with a peculiar and specific function.

We are thus drawn to the important conclusion that acute vesicular goitre has its own specific pathology and peculiar negative syndrome, and a high incidence in the male at puberty.

Special attention should be directed to this form of goitre from an experimental point of view, in that it is the antithesis of the adenoid goitre of primary Graves' disease. We see again, in these two conditions, that particular insistence on sex and puberty as an etiological factor to which little attention has been given in the experimental studies lavished on the thyroid apparatus. Gaskell's<sup>33</sup> phylogenetic studies of the thyroid gland are the only significant literature on this probable aspect of the thyroid function.

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\* Excepting, of course, occasionally pressure symptoms which are mechanical.

**Chronic Vesicular Goitre.**—The thyroid gland in this form of goitre has the appearance of a collection of colloid cysts surrounded by fibrous tissue. In this type of goitre, as in the acute type of vesicular goitre, the characteristic feature is the absence of any secretory tissue. *In the acute variety there is an active over-production of colloid, whereas in the chronic variety the accumulation of colloid appears to be produced by stagnation of the normal physiological turnover.* The incidence of chronic vesicular goitre in the sexes is not disproportionate when judged by operation material. How far this incidence is due to the fact that chronic vesicular goitre is also one of the types of endemic goitre is difficult to say, until such time as the survey of endemic goitre has been made from a pathological point of view.

Chronic colloid goitre is only one variety of endemic goitre. It presents a very different picture from the other form of endemic goitre to be dealt with later, and probably shows an independent etiology. We will return to a consideration of chronic colloid goitre after describing the second variety of endemic goitre.

#### DISORDERS INVOLVING BOTH COLLOID AND SECRETION.

We have dealt so far with the two extremes, in which either colloid is sacrificed to secretion, as in Graves' disease, or in which secretion is sacrificed to colloid, as in vesicular goitre. Clearly there must be a state of affairs in the thyroid gland wherein both these functions are exaggerated, constituting a true balanced hypertrophy.

It should be noted that hypertrophy is always a compensatory effort, and that the rule must hold that only the sequelæ of over-activity will lead to symptoms other than mechanical. In considering hypertrophic goitre it is sufficient to mention the physiological goitre of childhood, pregnancy, menstruation, and those of iodine medication and thyroid feeding. These physiological and pharmacological goitres are acquiring great significance because of the tendency to look upon all rises in the basal metabolic rate as indicative of pathological thyroid upset. The raised basal metabolic rate is, however, only another form of fever—apyrexial fever—and, as such, should take its place in diagnostics as indicating a necessity for compensatory effort somewhere in the metabolic cycle, but not necessarily in the thyroid, nor indeed necessarily pathological.

**Chronic Hypertrophic Goitre.**—We may turn to the consideration of one of the most important forms of hypertrophy in the thyroid gland, that of *chronic hypertrophic goitre*. This goitre is a true hypertrophy; overwork induces strain, and this results, as is usual, in a diffuse fibrosis throughout the gland. This fibrosis circumscribes areas of secretory hypertrophy (solid adenomatous tissue) and areas of excessive colloid storage (colloid cysts). In this country it is commonly called 'adeno-parenchymatous' goitre, and is one of the commonest forms of thyroid swelling.

Apart from its sporadic incidence, the importance of this goitre is that it constitutes the second variety of endemic goitres. It is undoubtedly the form of endemic goitre that was studied by McCarrison<sup>5</sup> in Gilgit, and it

coincides with one type of goitre prevalent in Switzerland and described by de Quervain<sup>34</sup> in that country. The etiology of the hypertrophy is still unknown.

#### ENDEMIC GOITRE.

It would appear from our studies that in the *adult* there are two varieties of goitre found endemically. In our opinion, one of the most valuable results of recognizing the existence of secretion as something apart from colloid-storage in the thyroid physiology is that it enables us to distinguish a chronic vesicular goitre from the chronic hypertrophic forms of goitre.

De Quervain (*see* Aschoff<sup>35</sup> also) in his recent work illustrates the two types of endemic goitre in patients of all ages. One type is undoubtedly the chronic vesicular goitre; the other variety, which he terms 'hyperplastic', corresponds histologically to our chronic hypertrophic goitre. He, however, draws no physiological distinction, as we do, between these two forms. The moment a physiological distinction is made, we have at hand a probable explanation of the very diverse results in the treatment of endemic goitre, and an explanation of the contradiction concerning the etiology of endemic goitre. It is improbable that the etiology of these two goitres can be the same; the correct treatment in each case will probably vary with the etiology.

There is a vogue for the indiscriminate treatment of endemic goitre with iodine. This is the revival of an old empiricism which no doubt lapsed for some good reason. The researches of Marine are largely responsible for the revival of iodine treatment. Marine maintains that iodine converts a 'hypertrophic' gland into a 'colloid' gland.<sup>31</sup> Chronic vesicular goitre, nevertheless, is undoubtedly the goitre that 'softens and dissolves', according to Kocher, under iodine medication. If Marine's contentions were correct, chronic vesicular goitre could only be aggravated by iodine therapy. McCarrison,<sup>5</sup> dealing with the chronic hypertrophic variety, finds the therapeutic administration of iodine useless. Indeed, in some zones, iodine appears to be a dangerous measure, as the figures of Fleischmann indicate.<sup>36</sup> He finds iodine may be toxic, and induce a so-called iodine-Basedowii (in Basel in 68 per cent of cases, in Berne in 28 per cent of cases, in Berlin in 37 per cent of cases). In Heidelberg, Krehl<sup>37</sup> finds iodine dangerous, and such authorities as Breuer, Möbius, and Ortner find a class of endemic goitre in which iodine is contra-indicated. (For a summary of these and other facts concerning iodine medication, *see* Falta.<sup>26</sup>) Whatever may be the effect of iodine in the goitre of children, we have said enough to indicate a need for the differentiation of endemic goitres before engaging in any specific treatment.

The basis of a differentiation appears to us to lie in the fact that vesicular goitre is functionally and etiologically different from goitrous swellings due to secretory hypertrophy (adeno-parenchymatous goitre), and that puberty and sex are important factors in their development.

In any case, it is a step forward to recognize *that there are two types of goitre with two distinct pathological expressions in endemic zones*, and, further, to recognize that these may be differentiated by the zone, or possibly by the sex in any one zone. In these considerations lie the grounds for a renewed attack on the problem of the etiology of endemic goitre.

## CONCLUSIONS.

I have summarized our particular conclusions at the end of each section. There remains only to mention the more general conclusions we may draw from this study.

Early Graves' disease and early acute vesicular goitre are apparently diseases of that function of the body over which the thyroid presides, and not, primarily, of the thyroid gland itself; whereas secondary Graves' disease, simple thyrotoxicosis, chronic vesicular goitre, and chronic hypertrophic goitre are primarily affections of the gland itself, and only incidentally and occasionally affect the functions of the body over which the gland presides.

It is necessary to seek out the precise function over which the thyroid apparatus presides if we are to unravel the complicated tangle of the thyroid problem. Had insulin (the endocrine secretion) been the first discovery in the study of the function of the pancreas, the precise position of the pancreas in relation to digestion might still be awaiting elucidation. We suggest there is a major function as well as an 'endocrine' one for the thyroid gland.

All that has been attempted in this paper is to present this problem anew to the clinician, and to indicate to him the various directions in which pathology points towards its solution.

We are indebted to Sir James Berry for the opportunity his material has afforded for this research work, and to him personally for his continued encouragement and help in the work. We have pleasure in acknowledging our gratitude to the Council of the Royal College of Surgeons for the opportunity of repeating many of our dissections in their laboratories, where the pervasive Hunterian atmosphere, and the help and encouraging criticism of Sir Arthur Keith, make labour light. We thank Dr. Hilda Cunningham for her continued help in the preparation of material.

Mr. Sewell's drawings speak for his patience and skill, and are an index of our considerable obligation to him.

This work is the substance of a report presented to the Medical Research Council. Personal association with Sir Walter Fletcher has been a real stimulus to work.

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## BONE-GRAFTING OF THE MANDIBLE: WITH REPORT OF SEVEN CASES.

BY WILLIAM BILLINGTON AND HAROLD ROUND, BIRMINGHAM.

FOR three years the writers, in association with Mr. Arthur H. Parrott, M.D.S., L.D.S., were in charge of the 'Jaw Centre' at the 1st Southern General Hospital, Birmingham, to which all cases of injuries to the jaws requiring special treatment were sent from the whole Southern Command. Upwards of 2000 soldiers were admitted to the Centre and treated by us. A very large percentage of these suffered from compound fractures of the mandible. Successful treatment involved: (1) Osseous union; (2) functional occlusion; and (3) Avoidance of disfigurement.

In a number of cases the fracture of the mandible was complicated by considerable loss of bone, either occurring at the time the wound was received or subsequently from necrosis, and a considerable gap existed between the fragments. It was found that if the gap exceeded half an inch, osseous union was rarely obtained unless (1) the fragments were allowed to approximate at the expense of normal alinement, or (2) a bone-graft was successfully introduced. In a number of cases the loss of bone was so extensive that any attempt to obtain union by approximation was impracticable. The need for a satisfactory method of securing union by means of grafts was very great, and much experimental work was carried out before a technique was arrived at which could be trusted to give uniformly good results. A description of the method adopted was published in the *British Medical Journal*, Dec. 21, 1918, and in the *Proceedings of the Royal Society of Medicine*, 1919, vol. xii (Section of Odontology, pp. 55 to 72). Altogether upwards of 75 cases of compound fracture of the mandible were successfully treated by means of bone-grafts, the gap varying in extent from half an inch to five inches. The longest graft employed was seven inches in length; it was successfully used in the case of a soldier whose lower jaw from angle to angle had been completely destroyed.

The knowledge gained by our experience of jaw-grafting in connection with war injuries has enabled us to remedy successfully the deformities and functional disabilities resulting from loss of a portion of the mandible as a result of the accidents and diseases of civil life.

The conditions for securing successful jaw-grafting are better in civil practice than they were in connection with war injuries. In the latter class of case we had to contend with extensive associated damage to soft tissues, and with virulent and prolonged sepsis, from which dense scars of low vitality resulted. These formed a very unsatisfactory bed into which to introduce the graft. As the result of our experience, we feel justified in claiming that, provided there is no active disease present, defects in the lower jaw can be

successfully remedied by bone-grafts, and firm osseous union obtained without loss of normal alinement in practically every case. The length of the gap between the fragments is immaterial. The technique is simple, but does involve close collaboration between surgeon and dental surgeon.

The mandible is peculiar in that success in grafting can rarely be obtained by the technique used in connection with the long bones. It is very intolerant of foreign bodies of any kind, and attempts to fix a free graft by wire, pegs, or plates almost invariably led to failure. Equally, fixation by dove-tailing the graft between the fragments was frequently unsuccessful. Further, fixation of the fragments by dental splints led to absorption of the graft if carried out before the operation or until the wound had firmly healed. After many experiments, all attempts to fix the graft or the fragments were abandoned, fixation only being secured by dental splints about a fortnight after the graft was introduced and the wound firmly healed.

Pedicle grafts have a very limited application, and are only of value when the gap is a comparatively small one. A further disadvantage entailed by their use is that the fragment from which the graft is taken is weakened and greater interference with the teeth is necessary. Owing to the ease with which free grafts can be introduced, and the uniformity of success that has attended their use, the employment of pedicle grafts has been entirely discontinued.

A matter of considerable importance is the choice of the bone from which to take the graft. Rib bone is too soft, and does not develop strength equal to that of the jaw generally. Grafts from the tibia are brittle, and cannot readily be bevelled and shaped to fit the gap; also, tibial grafts do not appear to accommodate themselves to their new environment, and are apt to necrose and work out. After experimenting with grafts taken from various parts of the body, the choice finally made was the crest of the ilium. The anterior superior spine and the crest as far back as necessary is freed from the attached muscles, and a graft as long as required is readily obtained by sawing through the bone below the crest from the anterior superior spine backwards. The advantages are that the graft is readily obtained and a minimum of injury inflicted—in no case has any subsequent disability been complained of. The bone is very tough and can be cut to any required shape by bone forceps without splintering, and the graft is thick and strong. Finally, grafts obtained in this way readily accommodate themselves to their new environment, and their nutrition rarely causes any anxiety. It is quite exceptional for any indication of absorption or rarefaction to be seen on X-ray examination between the time the graft is introduced and its firm osseous union with the fragments.

### OPERATIVE TECHNIQUE.

**Preliminary Preparation.**—It is absolutely essential that there should be no sepsis present in the field of operation. If this has existed, there should be an interval of several months after all signs of inflammation have subsided and the wound has firmly healed before a grafting operation is attempted. Unless this precaution is taken, there is risk of latent sepsis



lighting up. Great care should be taken to exclude the presence of sinuses opening into the mouth. Teeth immediately adjacent to the gap must be extracted, and all septic teeth or stumps in any part of the mouth removed. Just before the operation all splints and fixation apparatus should be removed, and no splints should be introduced for at least two weeks afterwards when the wound has healed. The presence of fixation splints within the mouth causes risk from post-anæsthetic vomiting, greatly adds to the discomfort of the patient, and, where pressure is exerted by them in or near the operation area, interferes with vitality and increases the risk of sepsis.

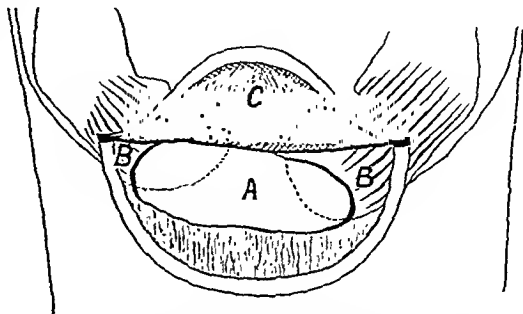


FIG. 312.—Diagram showing preparation of bed by raising a musculo-cutaneous flap (C). The graft (A) overlaps the bevelled ends of the jaw fragments (B B).

**Operation.**—A curved incision is made through the skin of the neck, beginning a full inch behind the extremity of the posterior fragment and ending a similar distance in front of the extremity of the anterior fragment (Figs. 312, 313). The incision commences and finishes about half an inch above the line of the lower border of the jaw and in the neck runs about an inch below that line. The incision is



FIG. 313.—To illustrate gap in mandible. The outer plate is removed from each fragment to the dotted line.

deepened by cutting upwards and inwards until the lower border of each fragment is reached. The soft tissues covering the outer surface of each fragment are then raised for an inch away from the gap and turned up in the flap. The ends of the fragments, together with the intervening fibrous tissue occupying the gap, are next carefully cut away. At this stage care is necessary to avoid making an opening into the mouth, an accident which necessitates aban-

donment of the operation and postponement until the wound has healed. The preparation of the bed is completed by removing a flake of bone from the outer surface of each fragment for an inch away from the gap. In doing this the exposed surfaces of raw bone are bevelled a little. All

bleeding is carefully arrested, and the wound covered up while the graft is being prepared.

The graft is taken from the same side of the body as the wound in the face. This ensures that the patient will be able to lie comfortably on one side when sleeping. An incision is made over the crest of the ilium, commencing at the anterior superior spine and extending as far back as required. The muscles are then separated on either side of the crest by cutting through their attachments with a scalpel as close to the bone as possible. The soft tissues are pressed back by ribbon retractors, and the bone is made to project for nearly an inch. The graft is cut with an ordinary Horsley hand-saw with movable back. The division of bone commences in front below the anterior superior spine, and is carried back until a graft is obtained which is two inches longer than the gap in the jaw. It is advisable to measure the length required on the crest and to make a vertical saw-cut at the required spot before commencing the horizontal division. This ensures that when the saw has travelled the required distance the graft becomes loose.

The detached piece of bone is held in a sterile mop, its extremities are bevelled with bone forceps, and any adhering soft tissue is trimmed away with scissors. The graft is then introduced into its bed in such a way that the bevelled ends overlap the gap by an inch at either end and lie on the prepared raw surfaces on the fragments (*see Fig. 312*). A little shaping is required to allow the graft to lie comfortably in place, but as the curve of the crest of the ilium approximates closely to that of the jaw, very little moulding is required. No attempt is made to secure fixation of the graft; it merely lies in the gap with its bevelled extremities extending over the fragments of jaw at either end. Two advantages result from the extensive overlapping: (1) A broad line of bony contact between the graft and the fragments is provided, with increased prospect of speedy, firm, osseous union; (2) There is practically no risk of separation in the event of the size of the gap being increased by the subsequent introduction of the dental fixation splints, as a certain amount of sliding can take place without contact being lost.

The soft tissues are sewn closely over the graft with chromicized catgut. This keeps it in place, has the additional advantage of surrounding the graft with living vascular tissue, and abolishes dead spaces in which blood-clot and serum can collect. Finally, the skin is approximated with a few interrupted stitches. No drainage is employed beyond that of leaving spaces between the skin sutures to allow of the escape of serum. A simple dressing and bandage is applied and the patient sent back to bed. The whole operation can be carried through deliberately and comfortably in about forty minutes. The patient complains of very little discomfort in the jaw, the chief complaint being directed to the wound in the hip.

No attempt is made to introduce the dental splints until the wound is firmly healed and the compound fracture has been converted into a simple one. This usually occurs in about two weeks, after which the case is treated as one of simple fracture of the jaw. Firm bony union occurs in from two to four months; but it is inadvisable to fit the final dentures until an interval of about six months from the operation.

**Dental Technique after the Operation.**—In from two to four weeks, according to the state of the wound and the patient, new models are taken and splints made for the fixation of the jaw in correct alignment. Any slight alteration in the occlusion, or any displacement of the fragments which may have resulted from the operation or in the interval prior to the re-adaptation of splints, is found to be easily reducible. This is possible owing to the fact that the graft, whilst being firmly held in position by the deep layer of soft tissues, overlaps the fragments at each end, and a certain amount of sliding can take place without bony contact being lost. The correction and retention of the fragments in their required positions is attained by the use of articulating splints, silver cap-splints adapted and cemented to any standing teeth, such splints being supplemented by vulcanite extensions. These extensions are lined with soft rubber where covering edentulous parts in the neighbourhood of the graft, in order that the parts may be retained in position with a minimum amount of pressure. The splints are fixed to each other by means of articulating tubes and bolts.

### PARTICULARS OF CASES.

*Case 1.*—Master F. S., age 11. In March, 1919, while playing with another boy on his father's farm, he was wounded by the accidental discharge of a loaded sporting gun. The left side of the face was severely lacerated, and the mandible between the left lateral incisor and a point just in front of the angle on the same side was completely destroyed. A preliminary plastic operation on the cheek and lower lip was performed by Dr. McLeod of Shrewsbury, and the right fragment of the mandible was maintained in good occlusion with the maxilla by a vulcanite splint having an inclined plane on the right side which caused the right fragment, on closure, to bite into normal position. We saw the patient in consultation in June, 1920. The wounds were firmly healed, but a wide gap existed in the mandible. It was decided to close the gap by grafting as the only means of securing union without great sacrifice of normal alignment.

Continuous metal cap-splints with articulating tubes were made for the maxilla and mandible. Considerable difficulty was experienced in their construction and fixation, both on account of the age of the patient and because there were very few teeth to use. The incisors in the maxilla had all been damaged, and the temporary teeth, most of which were carious and some loose, had to be used, the support obtainable from the permanent teeth not being sufficient.

In June, 1920, a grafting operation was performed. The bed was prepared in



FIG. 314.—*Case 1.* Radiograph of jaw six years after insertion of graft (side view). The dotted lines indicate the graft. A few spicules of lead are still present.

the usual way, and the graft taken from the crest of the left ilium. Part of the graft consisted of cartilage. The graft 'healed in' without complications, and three weeks after the operation the fixation splints were inserted in the mouth. A soft vulcanite block, fixed to the lower splint by means of vertical pins secured by vertical tubes on the left side of the lower splint, was inserted. This block had a horizontal tube fixed to its upper surface which articulated with two horizontal tubes on the upper splint. A pin passed through the three tubes bolted the upper and lower splints together, and at the same time held the block in position. This appliance remained fixed until October, 1920, when the splint became loose and was removed. Union was then found to have taken place at both ends of the graft, but as it was very 'springy' the splints were refixed and not removed until December, 1920, when good firm union was established. This was six months after the insertion of the graft. *Fig. 314* shows the jaw at the present time.

*Case 2.*—Mrs. B., age 34. In February, 1912, the late Mr. Jordan Lloyd excised two inches of the mandible for sarcoma. The patient was then 25 years old. Immediately after the operation one of us (H. R.) fixed the right fragment of the mandible into correct position with the upper teeth by means of interdental wiring,



*FIG. 315.*—*Case 2.* Radiograph of jaw taken four years after insertion of graft.

the upper and lower teeth being wired together. In four weeks the wires were removed and a restoration denture made. This and subsequent dentures were worn for nine years, when the patient was persuaded to submit to a bone-grafting operation.

In the interval between the two operations the patient had lost all her upper teeth, but she still had some teeth in the right fragment of the mandible: the left fragment was edentulous. The grafting operation took place in February, 1921, and two weeks later, when the wound had firmly healed, a vulcanite plate was made for the maxilla, and a metal cap-splint for the teeth on the right fragment. A movable soft vulcanite block attached to this articulated with

the vulcanite upper splint on the left side. The under surface of this block fitted that area in the mouth corresponding to the position of the bone-graft and extending on to the posterior fragment, so helping to steady this and the graft. All parts were fixed together by tubes and pins. A chin splint made of aluminium, and struck up on a model of the patient's chin, was worn; it was kept in place by elastic webbing passing over the scalp. This splint was necessary for a short while, because the upper plate, whilst being fixed to the lower splint, could not be fixed to the maxilla. The wearing of the chin splint quickly accustoms the patient to keep the mouth closed, and when this habit has been acquired the splint can be dispensed with.

Firm union had taken place at both ends of the graft three months after the operation. As a precautionary measure a restoration denture was worn for a further four weeks, making four months in all. *Fig. 315* shows the radiographic appearance four years after the operation.

*Case 3* (double bone-graft).—Pensioner Albert W., age 30. This man was admitted into the Queen's Hospital with an ununited fracture of the mandible resulting from war injury. There was a gap in the region of the first and second molar teeth on the left side. A bone-graft was inserted on Feb. 17, 1921, and

fixation splints were inserted on March 9. Good union took place, and the splints were removed on July 13, 1921.

He went to the United States, and in December, 1923, was assaulted and robbed by a gang of roughs. He was hit on the right side of the jaw with a piece of lead and the right side of the mandible was fractured. He was admitted into hospital



FIG. 316.—Case 3. Radiograph showing first graft four and a half years after its insertion.



FIG. 317.—Case 3. Radiograph taken four and a half years after insertion of graft on left side and one year after insertion of graft on right side.

in Philadelphia, and the fragments were wired. This was followed by suppuration and necrosis, and he was sent home to England with a discharging sinus and the wire *in situ*.

He was again admitted into the Queen's Hospital on Feb. 24, 1924. The wire and several sequestra were removed, and fixation splints applied in the hope of obtaining union. The wound healed, but there was no attempt at union. On July 1 a bone-graft was inserted. This healed in without trouble, and firm bony union had taken place in two months. The original graft had become firmly welded into the jaw, from which it could with difficulty be distinguished (Figs. 316, 317).

Case 4.—C. Evans, age 23. About seventeen years ago, at the age of 6, a portion of the right side of the mandible was removed by Mr. Leedham-Green for sarcoma. The fragments were kept in fair position for some years by bridging the six-year-old molar on the right fragment to teeth on the left fragment. Later the bridge became useless and the parts approximated considerably.

On April 5, 1922, a bone-graft was inserted, and good union resulted in four months. Fig. 318 shows the condition three years later.

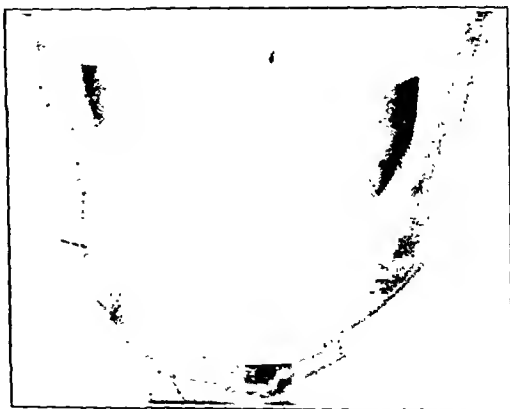


FIG. 318.—Case 4. Radiograph of jaw three years after insertion of graft.

*Case 5.*—Florence Parker, age 8. This child suffered from severe osteomyelitis of the mandible when 5 years old. Considerable necrosis occurred, and a gap in the continuity of the mandible resulted. The gap was situated just in front of the



FIG. 319.—*Case 5.* Radiograph of jaw three and a quarter years after insertion of graft. An unerupted tooth is shown in the posterior fragment.

third molars were found malplaced and unerupted. The teeth lay horizontally in the jaw in the region of the angle, with their crowns in juxtaposition and locked together. The roots of the third molar extended backwards into the ascending ramus, and those of the second molar downwards and forwards just in front of the angle. Both teeth were placed centrally to the jaw, and the surrounding bone was extremely thin and dense.

With considerable difficulty the teeth were removed in November, 1923, but the bone was so thin and brittle that a fracture occurred. The fragments were kept in as good alinement as possible by inter-maxillary dental wiring until the wound had healed sufficiently to allow fixation of splints. The vitality of the bone at the site of the fracture was so poor that absorption took place.

As there was no indication of union at the end of four months, it was decided to insert a bone-graft. The operation was performed on March 23, 1924. Uninterrupted recovery took place, and on July 5, 1924, firm union had occurred and the splints were removed.

*Fig. 320* shows the condition one and a quarter years after the operation.

*Case 7.*—Mrs. H. In January, 1924, the patient attended the Queen's Hospital complaining of pains in the right arm. For several months the pains were regarded as rheumatic, but as they became more severe in spite of treatment, the jaw was radiographed, and what appeared to be an odontome in the region of the second

angle on the right side. A graft was inserted in April, 1922. The graft, which was composed largely of cartilage, was taken from the crest of the ilium. Some doubt was felt whether a graft mainly composed of unossified cartilage would be a success. This doubt was intensified by the fact that within a week of the insertion of the graft the child developed scarlet fever, and she had to be sent away to a fever hospital without fixation splints. These could not be applied for over a month. Firm union was, however, obtained four months after the operation. *Fig. 319* shows the result after three and a quarter years.

*Case 6.*—Mrs. B. For twelve years this patient had suffered from attacks of neuralgia in the right side of the head. These attacks became increasingly worse. On X-ray examination, the second and



FIG. 320.—*Case 6.* Radiograph of jaw one and a quarter years after insertion of graft.

bicuspid was shown. As the supposed odontome involved almost the entire depth of the jaw, it was decided to excise the section of the mandible in which it was situated. This was done, and it was then found that the trouble was caused by an unerupted second bicuspid which lay horizontally with its root incorporated in very dense bone. This obscured the root in the radiograph, the crown alone being

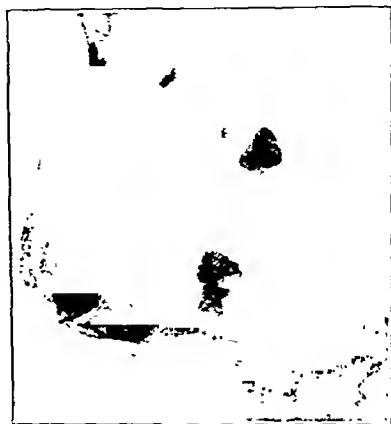


FIG. 321.—Case 7. Radiograph of jaw nine months after insertion of graft.

discernible. In September, 1924, the gap was closed by a bone-graft. Union took place rather slowly at the anterior end of the graft, but in February, 1925, it was satisfactory, and the splints were removed. The arm pains completely vanished after the operation in June. Fig. 321 shows a radiograph taken in June, 1925.

We desire to acknowledge the valuable help we have received from Dr. Brailsford in connection with the radiographs.

## COMPLETE EXPOSURE OF THE RADIUS.\*

BY ARNOLD K. HENRY, CAIRO.

THE entire length of the radius is most easily exposed through an antero-lateral incision.

## ANATOMY.

Three long muscles flank the bone upon its outer or lateral border—the brachioradialis, miscalled the supinator longus, and the long and short radial extensors of the wrist (*Fig. 324*). They can be retracted outwards when they have been relaxed by bending the elbow. These three muscles have origins which are proximal to the elbow-joint, and in order to mobilize them fully, the incision must extend proximal to the elbow. Further, this lateral group of three is strapped to the lower fourth of the radial shaft by two small muscles—which form an oblique prominence when the hand is prone. When the long muscles are raised from the bone these two short muscles are raised with them. The *pronator teres*, inserted at the middle of the radial shaft upon its outer face, will then be exposed, and full pronation of the hand will reveal an expanse of bone in the lower half of the shaft.



FIG. 322.—Anatomical relationships at the upper part of the radius. The white crescent between the two black insertions represents the bicipital bursa and shows how it lies in a bay formed by the supinator edge. The surgeon is guided to the bursa by the outer face of the biceps tendon.

The *supinator muscle* grasps the upper third of the shaft, and the *posterior interosseous nerve* (deep branch of the radial—B.N.A.) penetrates the anterolateral face of the supinator, and lies between two layers of the muscle: the deep layer separates the nerve from the bone. The supinator attachment to the radius skirts the edge of the bicipital tuberosity, which is covered in front by a bursa (*Fig. 322*).

*The Deep Guide.*—The edge of the supinator is obscured by the loose connective tissue that surrounds the recurrent radial vessels, but it can be reached with precision by following the outer face of the *biceps tendon*. The outer face of the tendon leads the surgeon to the bursa: the knife cuts through this to the tuberosity and at once affords the rugine a point of direct contact with the bone (*Fig. 325*). It is

\* Shown at the Section of Surgery, Royal Academy of Medicine in Ireland, April 3, 1925.



then easy, starting at the edge of the muscle, to begin detaching the grasp of the supinator from the radius. The biceps tendon is thus the deep guide in this exposure, and the surgeon must keep strictly to its outer side or he may injure the ulnar vessels.

The recurrent radial vessels curve outward at the level of the bicipital tuberosity, and can be hooked up on the finger and divided. The finger then gains admission to the plane of cleavage between the pronator teres and the brachioradialis (*Fig. 324*). The anatomy of this plane favours the surgeon, for the radial artery runs within the sheaths of the pronator teres and the flexor longus pollicis, while the radial nerve (superficial branch of the radial—B.N.A.) is attached to the long lateral muscles which flank the radius. The separation of these neighbouring structures is thus made easy.

### THE OPERATION.

*Incision (Fig. 323).*—With the patient's elbow extended and the hand supine, divide the skin and superficial fascia by a straight incision from a point at the outer edge of the biceps belly four finger-breadths proximal to the bend of the elbow, and terminating at the tip of the radial styloid. Divide and tie the large superficial vein which crosses the mid-third of the radius.



FIG. 323.—Incision for exposure of the radius. Divide skin and superficial fascia from the lower end of the biceps belly to the radial styloid. Begin dividing the deep fascia over the biceps tendon.

*The Deep Guide.*—First expose the biceps tendon by dividing the deep fascia at its outer side. Continue the division of the fascia throughout the wound with blunt-nosed scissors. Pass the

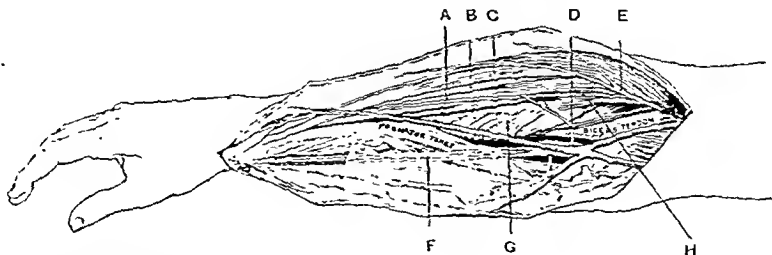


FIG. 324.—Exposure of the biceps tendon: slide a finger downwards along its outer side till it meets the resistance of the recurrent vascular loop. Divide and tie this loop. Mobilize the three long muscles which flank the radius: flex the elbow and retract them outward. A, Extensor carpi radialis brevis; B, Extensor carpi radialis longior; C, Brachioradialis; D, Recurrent vascular loop; E, Musculocutaneous nerve; F, Radial artery; G, Supinator; H, Posterior interosseous and radial nerves.

finger distally along the outer side of the tendon till it meets the resistance of the recurrent vascular loop (*Fig. 324*). Hook this up gently on the finger: divide and tie it. Mobilize the three long muscles which flank the outer

face of the forearm. Detach the flat tendon of the brachioradialis from its insertion into the base of the radial styloid. Flex the elbow through  $90^\circ$ , and retract the muscles widely out to expose the supinator.

Return to the biceps tendon: keep the knife close to its outer face and cut down upon the radial tuberosity (*Fig. 325*). This cut divides the

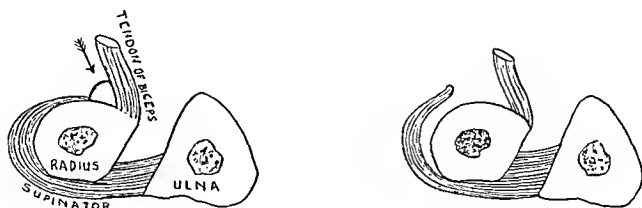


FIG. 325.—Showing the cut made through the bicipital bursa. Keep the knife close to the outer face of the biceps tendon. The supinator is then peeled off the radius. The arrow shows the direction of the cut through the bursa.

bicipital bursa, and the knife strikes the radial tuberosity where it lies in a bay formed by the supinator edge (*Fig. 322*). From this strategic point the rugine peels the supinator muscle off the bone. The muscle is turned out-

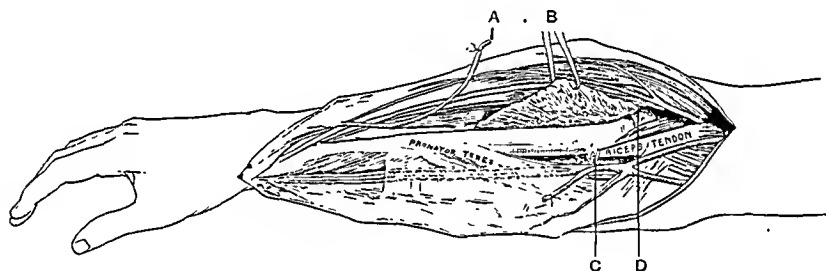


FIG. 326.—Completion of the exposure by putting the forearm in full pronation. A, Radial vein; B, Foreeps retracting reflected supinator; C, Recurrent vascular loop (cut); D, Posterior interosseous nerve.

wards, carrying within its substance the posterior interosseous nerve. Put the forearm into a position of full pronation, and the radius will be revealed from end to end (*Fig. 326*).

My best thanks are due to Professor E. J. Evatt, D.S.O., for the opportunity of working out the details of this exposure in the School of Anatomy, Royal College of Surgeons, Ireland, and to Mr. Matthew Barry for his drawings.

# DESQUAMATIVE AND DYSGENETIC\* EPITHELIAL HYPERPLASIAS IN THE BREAST:

## THEIR SITUATION AND CHARACTERISTICS: THEIR LIKENESS TO LESIONS INDUCED BY TAR.

BY SIR GEORGE LENTHAL CHEATLE, LONDON.

SOME critics have declared that they can attach no importance to my observations upon desquamative epithelial hyperplasia in breasts, for two reasons: first, that it so frequently occurs in the breasts of women who have died of maladies unconnected with these glands; and, second, that it is also commonly seen in breasts containing other swellings which have been removed from living women. The second reason seems to me to contradict the importance my critics give to the first, for the second reason suggests what I believe to be true, viz., that there is a definite though distant connection between desquamative epithelial hyperplasia and the genesis of swellings that occur in the breasts of living women.

These facts are common knowledge, but I do not agree that because desquamative epithelial hyperplasia of the breast is so commonly seen in these circumstances, its presence is therefore unimportant. On the contrary, I believe it to be a pathological state within the actual lesions of which many tumour formations begin. The situation which desquamative epithelial hyperplasia occupies, the pathological changes that occur in it, the great length of time it persists, and the genesis of these additional changes therein, make its presence one of menace and importance. The site of its incidence is important, because it is situated mainly in the acini and terminal ducts, where also occur many other forms of epithelial hyperplasia, including a type of carcinoma of a very definite character, all of which I shall describe in this paper.

The importance of the time factor is this: Carcinoma beginning in a part where irritation is present, or even where irritation has been applied, may take a long time to mature. The period necessary for the induction of tar carcinoma in human beings is from twenty to thirty years. In connection with this fact, there are two more facts to which I must draw attention: (1) Desquamation of epithelium, papillomata, a type of dysgenetic epithelial

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\* I substitute the term 'dysgenetic epithelial hyperplasia' for that of 'non-desquamative epithelial hyperplasia', a term I have used hitherto. By 'dysgenetic epithelial hyperplasia' I mean one that results in the formation of epithelial cells that are growing pathologically, not one that is throwing off cells that probably are of no further importance, i.e., 'desquamative epithelial hyperplasia'. The term includes: (1) A state wherein the growing epithelial cells show nuclear hyperchromatosis, variation in size, and mitoses (see *Slides A and B, Class II*); and (2) One in which these cell changes are not present (papillomata).

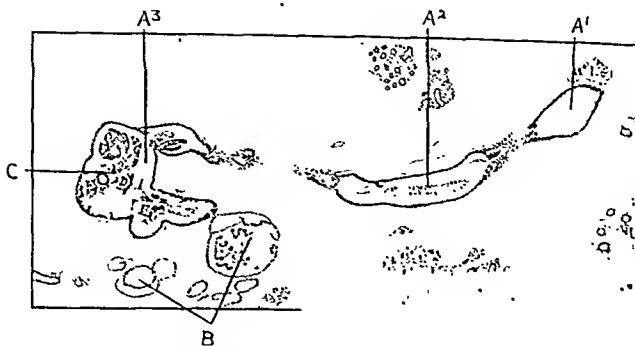


FIG. 327.—Part of whole section removed from female, age 34, for localized pain and nodularity of the lower and outer portion of the breast.  $A^1$ ,  $A^2$ ,  $A^3$  is the termination of a duct, B the acini into which it led. At  $A^2$  desquamative epithelial hyperplasia is present, resulting in a collection of colostrum-like cells. At C in  $A^3$  there is a multiradicular papilloma. At B desquamative epithelial hyperplasia of the acini has created a small cystic state.

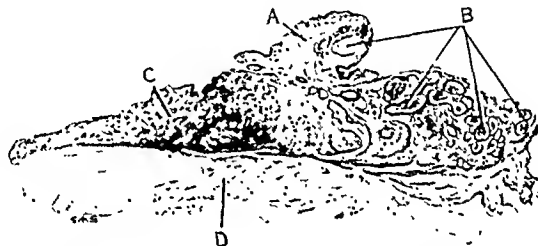


FIG. 328.—Whole section of breast removed from patient, age 41. A, Nipple. D, Pectoralis major. C, Normal portion of the breast. B, The only dilated duct in the breast. Its terminal branches are filled with the papillomatous tumours seen in Fig. 329. These terminal branches lead into acini dilated by sessile non-papillomatous dysgenetic epithelial hyperplasia. There is no epithelial-cell invasion in this breast. It is a case of my own, and my clinical diagnosis was carcinoma. There were no axillary lymphatic glands affected.

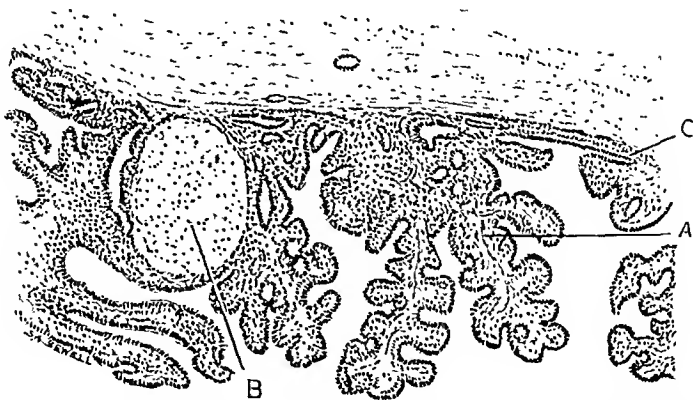


FIG. 329.—Taken from a duct of Fig. 328. C, Normal epithelial covering of the duct wall from which grows the papillomatous state seen at A, and which resembles the state of warts that occurs on the skin after the application of tar. B, Collection of colostrum-like cells that can be seen growing from the epithelial wall of the duct. In other parts of this duct the epithelial growth is sessile. The cells there show nuclear hyperchromatosis, mitosis, and they are irregular in size.

hyperplasia that does not invade outside tissues although histologically malignant, and carcinoma, all follow one after the other in this sequence before tar carcinoma appears in human beings (*see Figs. 342, 343, and 344*); (2) In the breast this sequence of events is precisely the same as that which I have just given in connection with tar cancer—viz., desquamative epithelial hyperplasia, followed by papilloma formation, which is in turn followed by a form of epithelial hyperplasia that is histologically malignant, but which in the absence of epithelial invasion cannot be said to be malignant (*State A, Class II*), and finally carcinoma (*see Figs. 327-340, 345, 347-349, 351-353*). These events can be seen occurring frequently in the lesion of desquamative epithelial hyperplasia. In producing these facts I have done enough to place desquamative epithelial hyperplasia in the position of initial importance.

It would not be correct to say that I propose excision as the only remedy. My observations entitle me to say only that many years may pass before more important and various pathological changes are induced in the lesions of desquamative epithelial hyperplasia, and that in any of these pathological changes, when once induced, intercurrent impulses may arise that arrest or prolong them, or hasten them into carcinoma.

However, I have described other changes that are not to be discovered frequently in the breasts of women who have died of other diseases; in fact their detection in post-mortem breasts is decidedly uncommon. As it is to these changes I wish to draw more particular attention, an epitome of my observations that bear on all relevant pathological changes (including desquamative epithelial hyperplasia) will enable me to make my demonstration clear to my readers.

In this paper I divide these pathological conditions into two classes:—

*Class I.*—Desquamative epithelial hyperplasia, i.e., an epithelial hyperplasia that results in a collection, within acini and terminal ducts, of cast-off epithelial cells which are probably of no danger to the individual. *Class I* contains those forms of epithelial hyperplasia to which I alluded in the first paragraph of this paper.

*Class II.*—Dysgenetic epithelial hyperplasia, i.e., one that results in a collection of living and pathological epithelial cells which may lead to three separate conditions:—

1. A condition in which the epithelial cells retain their normal structure; it produces papillomata confined within the acini and terminal ducts.

2. An epithelial hyperplasia in which the individual cells are showing signs of beginning malignancy—e.g., nuclear hyperchromatosis, variation in the size of cells, and mitosis—yet the process is still confined within acini

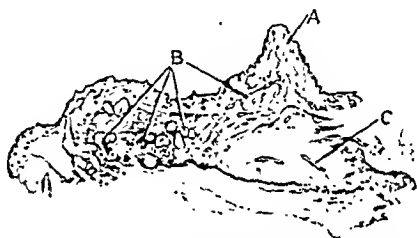


FIG. 330.—Whole section of a breast removed from female, age 36, for irregular nodularity in the lower and outer part of breast. Compare the whole section of Fig. 328, which belongs to the same type of pathological change. A, Nipple. C, Normal portion of the breast. B, Part of the only dilated duct in the breast. The terminal branches of it contained papillomatous multiple multiradicular tumours (*see Figs. 331, 332, and 333*). The terminal ducts led into many acini that were distended by non-papillomatous dysgenetic epithelial hyperplasia.

and terminal ducts and has not extended into the surrounding connective tissue (*State A, Class II*).

3. A more advanced condition in which, in addition to the cell changes, the process has extended beyond the walls of the acini and terminal ducts and has invaded the surrounding connective tissue (*State B, Class II*).

The presence of one or more varieties of dysgenetic epithelial hyperplasia in the same specimens in which early or late carcinoma is present is most significant (see *Figs. 346-355*).

All the specimens I reproduce as examples of these two classes were taken from living women. To *Class I* belong those changes that are so frequently discovered in breasts of women who die of maladies unconnected with breast

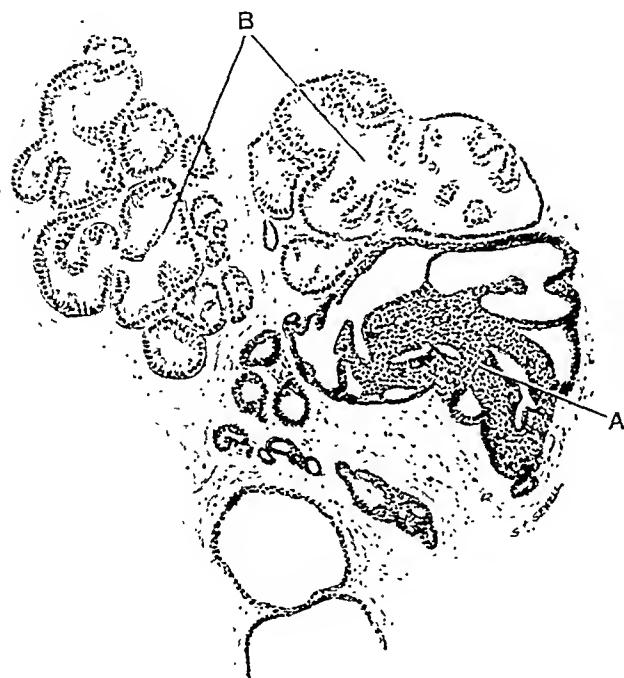


FIG. 331.—Taken from terminal duct and acini of Fig. 330. In the last portion of a terminal duct at A there is a multiradicular papillomatous tumour. This leads into the acini B, which are in an early cystic state.

disease. Although the specimens that belong to *Class II* have been removed from living women, these women might have died from maladies unconnected with breast disease while the lesions I reproduce here were present. Hence the occasional post-mortem discovery of these lesions in such circumstances ought not to create surprise, nor should it lessen their importance from pathological and practical standpoints.

#### *Class I.*—DESQUAMATIVE EPITHELIAL HYPERPLASIA.

Of this there are two types, *A* and *B*.

*Type A.*—Less common than *Type B*. In it there is a more or less completely generalized collection of desiccated, irregularly-shaped, ill-staining cast-off epithelial cells. The process is situated in the acini and terminal

FIG. 332.—Another terminal duct from *Fig. 330*, and contains the same type of tumour. Growing from the wall of the duct can be seen a desquamative type of epithelial hyperplasia (A) resulting in the collection of colostrum-like corpuscles.

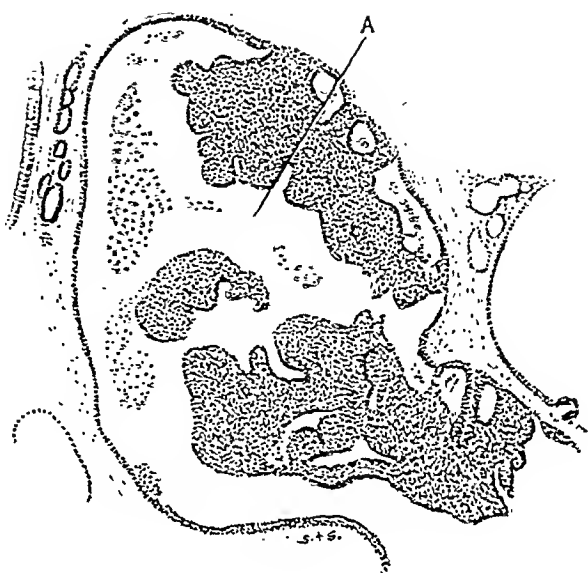


FIG. 333.—Drawing made from another terminal duct of *Fig. 330*. C, The papillomatous tumour it contained; A, Normal epithelium lining the duct; B, Histological appearances that suggest epithelial cell invasion. Among these cells are some showing nuclear hyperchromatism, mitosis, and irregularity in shape.

ducts. This type may remain many years without alteration in histological appearances. It is seen in a great many fibro-adenomata.

*Type B.*—Much more frequent and important than *Type A*. The process affects terminal ducts alone, acini alone, or it may appear as a combined affection of acini and terminal ducts (*see Fig. 327*). All varieties of *Type B* may be present in the same breast. In so far as it affects terminal ducts and acini, it resembles in situation *Type A* and the types of dysgenetic epithelial hyperplasia described in *Class II*. *Type B* is often a much more localized affection, and I feel sure it is much more important than *Type A*, because many forms of epithelial hyperplasia complicate the actual lesion it creates. For example, *see Fig. 327*, where a terminal duct affected by *Type B* is represented. In it, there is a multiradicular papilloma that was clinically undetectable. (*See also Figs. 328–340.*)



FIG. 334.—Papillomatous tumour in a duct which leads directly into the terminal ducts and acini seen in *Fig. 335*. *Figs. 334* and *335* illustrate the only lesions to be discovered in the breast, after cutting whole sections. There was Paget's disease of the nipple.

*Type B* is easily recognized. In the early stages in the terminal ducts and acini, the epithelium undergoes hyperplasia and becomes elongated and feathery in appearance. The terminal ducts can often be traced to only one main duct, which may be the only duct affected in the whole breast (*see Figs. 328* and *330*). Only in the ducts the desquamated epithelial cells appear like colostrum corpuscles, which can be seen in all stages of formation arising from among the feathery cells. When the affected ducts are larger than those that are terminal, the feathery character of the epithelial hyperplasia is absent, but the colostrum-like cells can be seen in all stages of formation arising from the ordinary cells that line the large ducts. On rare occasions the desquamated cells are seen to have separated parts of the normal walls of ducts, through which they are being pushed into the tissues immediately outside the ducts (*see Fig. 341*). From these particular cells probably no other evil need be anticipated. It is in the



lesions of *Type B* that the genesis occurs of the dysgenetic epithelial hyperplasias I shall presently describe.

*Type B* is specially a cause of duct and acinous cysts. I have pathological and clinical evidence to adduce which shows conclusively that suspected and unsuspected papillomata and carcinomata respectively, or both combined, occur in these cysts.

Both *Types A* and *B* are frequently accompanied by hyperplasia of the connective tissue that immediately surrounds the ducts and acini. Whether

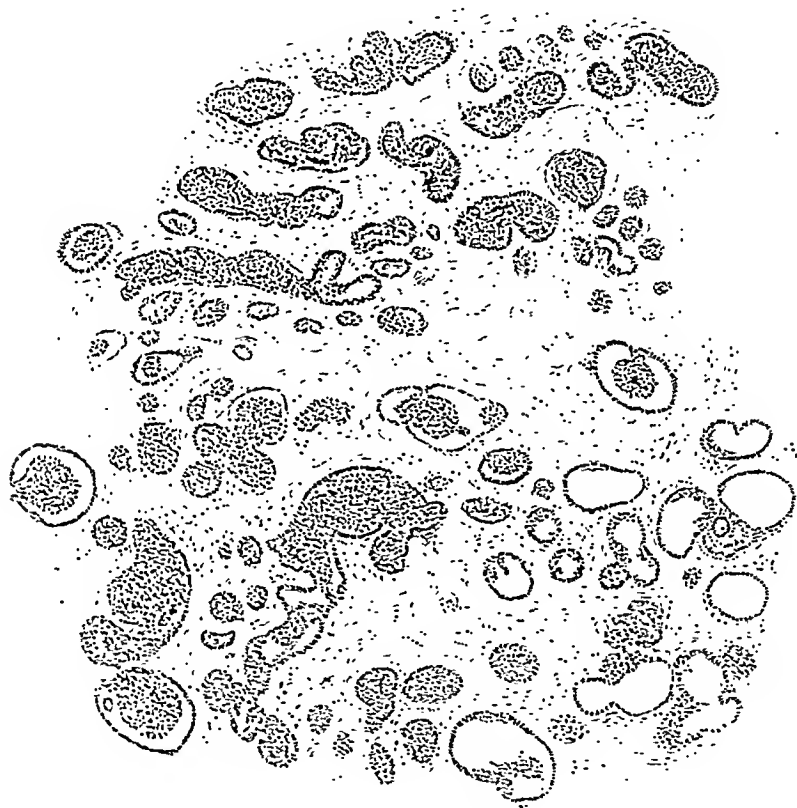


FIG. 333.—Termination of terminal duct of Fig. 331. In the ducts can be seen a mixture of papillomatous and sessile dysgenetic epithelial hyperplasia. Most of the acini are filled by an actively staining epithelial hyperplasia. There is no transgression of natural boundaries. Among the epithelial cells are mitotic figures, nuclear hyperchromatosis, and variation in shape.

this hyperplasia of the connective tissue is due to the same inducing agent as that of the epithelial hyperplasia, or whether it is only a response to the mechanical stretching induced by the dilating glandular elements, it is impossible to say. The pathological changes in *Class I* (specially *Type B* of



FIG. 336.—Section removed from lower part of breast of female, age 41. The provisional clinical diagnosis of a fibro-adenoma was made. Within the marked areas, at 1, 2, and 3, separate collections of terminal ducts and acini are filled by dysgenetic epithelial hyperplasia, which, in the ducts, is papillomatous. The only transgression of natural boundaries that occurred in this specimen was that the acinous cysts that were filled by epithelial cells were becoming confluent. Whole sections made of this breast revealed the fact that these terminal ducts led into one main duct, which was the only part of the breast affected. A, A<sub>2</sub>, B<sub>1</sub>, B<sub>2</sub>, B<sub>3</sub>, B<sub>4</sub>, see text, p. 529.



FIG. 337.—Breast of a woman, age 52. There was Paget's disease of three months' duration on the top of the nipple. A, Part from which Fig. 338 was taken.

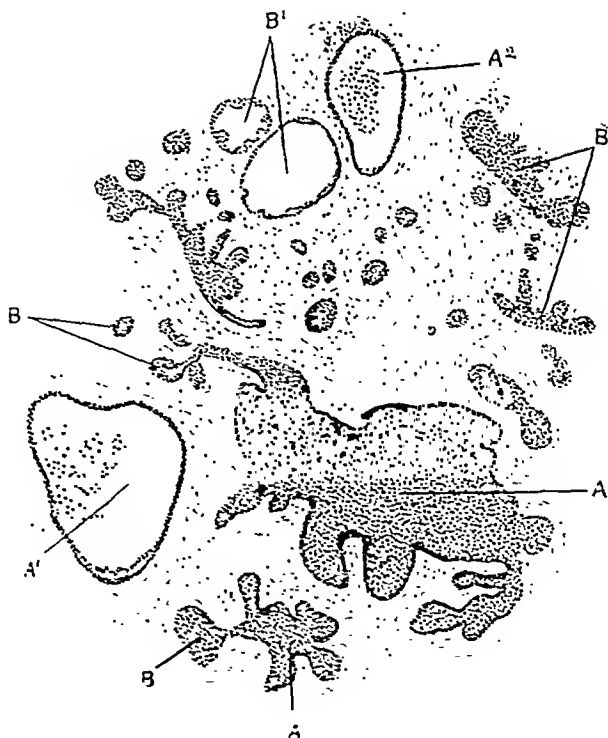


FIG. 338.—The only part of the breast in Fig. 337 that showed any dysgenetic epithelial hyperplasia. The terminal ducts are filled with papillomatous epithelial hyperplasia, and the acini are filled by growth that is not papillomatous. There was no transgression of normal boundaries.



FIG. 339.—Whole section of a breast, from which Mr. G. L. Keynes allowed me to cut this section. I did not examine the whole breast. At A is the only part in the portion of the breast I received in which I could find dysgenetic epithelial hyperplasia, seen in Fig. 340.

FIG. 340.—From A in Fig. 339. One terminal duct and its branches (A) were affected by sessile dysgenetic epithelial hyperplasia, in which nuclear hyperchromatosis, mitoses and irregularity of shape are marked. B represents the same type of hyperplasia in the acini. There was no invasion by epithelial cells in this specimen. The duct A<sup>2</sup>, which is filled by colostrum-like cells as the result of epithelial hyperplasia, leads into the acini, B<sup>1</sup>, which are in the early state of cystic formation. A<sup>1</sup> is another portion of a terminal duct which is undergoing the same process as seen in A<sup>2</sup>.



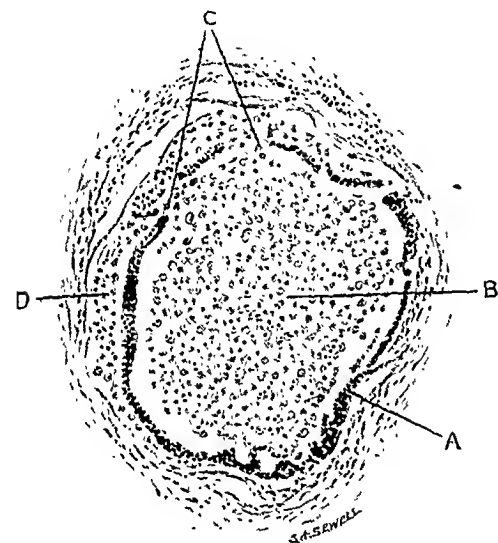
that class) can be identified as playing definite preliminary parts in the sequence of events included in *Class II*, and it is thereby rendered a class of pathological and practical importance.

This sequence of events, beginning in epithelial hyperplasia that desquamates, passing on to epithelial hyperplasia that does not desquamate, but which gives rise to living pathological epithelial hyperplasia that may end in carcinoma, is beautifully brought out by *Figs. 342, 343, and 344*, which have been kindly lent to me by Dr. J. A. Murray, and represent the results, at different stages, of the application of tar to the skin of animals.

Take *Fig. 342*. Here the desquamating process is shown after a few applications of tar to the skin; horny layers of the epidermis are being piled up on one another as the result of desquamative hyperplasia of the superficial epithelial layers; the deepest layer is inactive and strictly limited to its normal boundaries. The behaviour of this skin corresponds to that of desquamating processes of the breast in *Fig. 327, A*, and *Fig. 341*.

The second lesion in sequence is the formation of warts, and is dysgenetic in type. Epithelial cells removed from these warts and placed under the skin of the same animal either atrophy or form implantation cysts. These warts correspond to the formation of papillomata in the breast.

*Fig. 343* represents the third of Dr. Murray's experiments. The epithelial hyperplasia is also dysgenetic in type, and is a result of a longer application of tar to the skin. Living epithelial cells are being accumulated which are capable of dangerous activity, as proved by the fact that Dr. Murray placed



*FIG. 341.*—Transverse section of a duct the epithelial cells lining which are undergoing desquamative epithelial hyperplasia. A, Wall of the duct. B, Colostrum-like corpuscles filling and distending the duct. At C the epithelial wall has given way, and these colostrum-like corpuscles can be seen emerging from the duct and occupying the surrounding tissue outside it (D).

some of them under the skin of the animal, where they grew and metastasized. Yet the deepest layer of epidermis remains sacred to its normal boundaries. This condition corresponds to *State A, Class II*, in the breast.

The fourth experiment of Dr. Murray, shown in *Fig. 344*, is so obvious that no further allusion to it is necessary, except to demonstrate the final catastrophe of carcinoma.

The changes observed in Dr. Murray's experiments and also those that occur on human skin constantly exposed to tar are identical in sequence and comparative duration to those processes in the breast included in *Classes I and II (Figs. 327 and 341 of Class I, also Figs. 328-355 of*



FIG. 342.—Skin of animal after few applications of tar. There is only a desquamative epithelial hyperplasia at A. (Dr. J. A. Murray kindly gave me leave to publish this and the two following pictures.)

FIG. 343.—Skin of another animal after more applications of tar than those administered to specimen in Fig. 342. The epithelial hyperplasia is dysgenetic, and cells taken from A were placed under the animal's skin, where they grew and metastasized. B shows that the normal boundaries of the epidermis have not been transgressed. (Dr. J. A. Murray's experiment.)

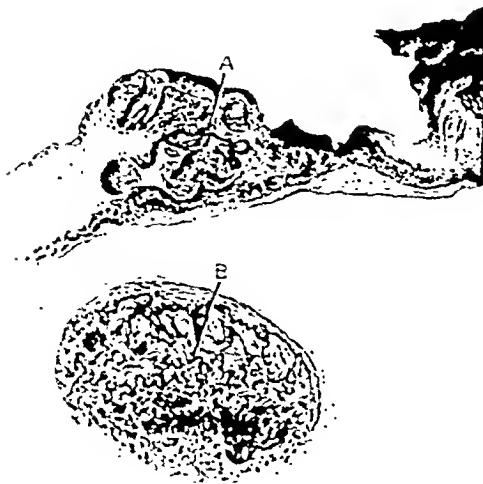


FIG. 344.—Skin of another animal after still more applications of tar. Carcinoma exists at A. B is a lymphatic gland enlarged and filled with epithelial cells from A. (Dr. J. A. Murray's experiment.)

*Class II*). The desquamated cells in the first of his experiments are thrown off from the skin surface into the air. Desquamated epithelial cells of the

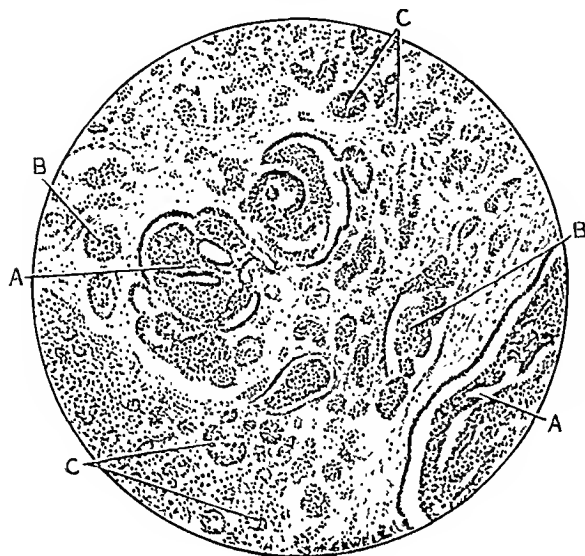


FIG. 345.—Section of a terminal duct (A), and some of its acini (B). At C epithelial cells have invaded structures outside the ducts and acini. The dysgenetic epithelial hyperplasia in the duct A is papillomatous. The specimen is taken from a breast of a female, age 42, who, eight years before its removal, had complained of pain in the affected breast. Four years before its removal, there was a discharge of blood from the nipple, the only occasion on which blood had been noticed. A few months before its removal, the breast became more painful. There were small multiradicular papillomata beginning in the ampulla and occupying the whole duct and its terminal branches. It was the only duct affected. The growth in the acini is not papillomatous. There were no lymphatic glands enlarged in the axilla. (Dr. W. R. Smith's patient.)

breast must collect in and distend the affected ducts and acini (see Figs. 327 and 341). The third of Dr. Murray's experiments (see Fig. 343) is the

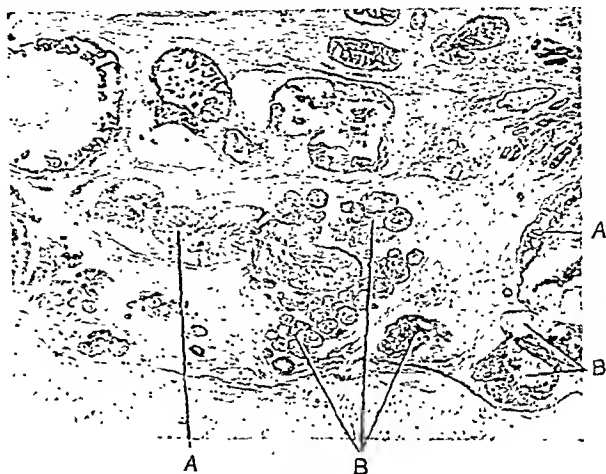
FIG. 346.—Whole section of breast removed from woman, age 47. Half of the section is occupied by a carcinoma (A). The other half (C) is normal except for a few small cysts just outside the periphery of the photograph. B, Nipple. D, Pectoralis major. There were large discrete lymphatic glands in the axilla containing epithelial cells. The woman died two years after removal of the breast and axillary lymphatic glands. The main size of the tumour was due to the enormous dysgenetic epithelial hyperplasia that was contained within dilated ducts and acini (see Figs. 347 and 348). There was marked epithelial cell invasion among the tissue surrounding the affected ducts and acini.



most deeply interesting of the four. As I have already pointed out, he took away some of the cells from their position at A, and placed them under the skin of the animal, where they grew and metastasized. Is it to be inferred that those dysgenetic epithelial cells, in their undisturbed position

at A, were malignant, or did they become malignant only when they were removed and inserted into tissues outside the epidermis? The importance of this point lies in the fact that a dysgenetic epithelial hyperplasia in the

FIG. 347.—Photograph taken by Mr. Walter Barnard of the edge of the tumour in Fig. 346. The ducts A, A were surrounded by normal elastica, and led into the acini B, B. The dysgenetic epithelial hyperplasia in all these structures was non-papillomatous. The epithelial hyperplasia is contained within normal boundaries.



breast gives rise to a most suspicious-looking collection of living cells, while they are strictly confined within ducts and acini (see *State A, Class II*).

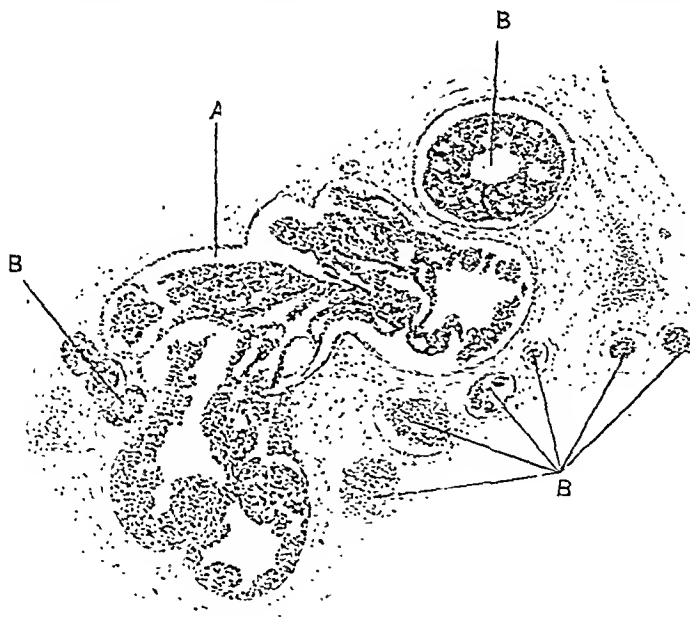


FIG. 348.—Drawing of acini and terminal ducts made from the centre of the tumour in Fig. 346. The elastica of the duct A was intact. The dysgenetic epithelial hyperplasia in the duct is papillomatous. The growth in the acini B, B, B is not papillomatous. Like the epithelial hyperplasia in Fig. 347, it is strictly confined to normal boundaries, although taken from the carcinomatous tumour in Fig. 346.

Here the following question arises. Should these dysgenetic epithelial cells of the breast gain admission into outside tissues by traumatic, chemical, or some other means, would they be capable of multiplication and metastasis? I believe they would behave in this way when they appear active and not degenerating, and, in spite of an absence of epithelial-cell invasion, I consider them to be potentially malignant, a belief that is supported by the fact that



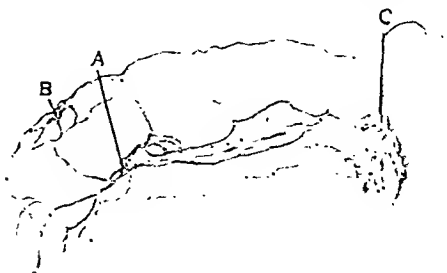
FIG. 349.—Drawing made from the centre of a carcinomatous tumour in the breast of a woman, age 51. 1, 2, Terminal branches of the same duct, surrounded by clastica. 4, 5, Acini. 3, Area in which epithelial cells have invaded surrounding structures. The dysgenetic epithelial hyperplasia in the duct (1, 2) is papillematous. The growth in the acini is not papillomatous. The lymphatic glands in the axilla contained carcinoma, and the patient died four and a half years after the operation. The epithelial cells in the duct show nuclear hyperchromatosis, mitosis, and irregularity in shape, corresponding thereby to the epithelial cells in Figs. 328, 330, 333, 334, 340.

precisely similar histological appearances are seen in the ducts and acini of tumours where epithelial cells have escaped from their normal boundaries and constitute a state of carcinoma (*see* the ducts and acini in carcinomatous tumours in Figs. 345–354).



I admit that at present it is impossible to determine absolutely whether a microscopical preparation demonstrates the presence of carcinoma, unless it shows a dysgenetic epithelial hyperplasia from which epithelial cells have

FIG. 350.—Drawing from a whole section of half a breast that was removed twenty years ago for the purposes of diagnosis. The woman was 50 years old, and is still alive. The tumour (A) is the smallest carcinoma I have removed from a breast. C, Position of the nipple. B, A small portion of skin covering the gland. There were no axillary glands affected.



invaded surrounding structures, where they multiply and undergo metastasis. Yet in the earliest stages of the carcinomatous process there must be one in

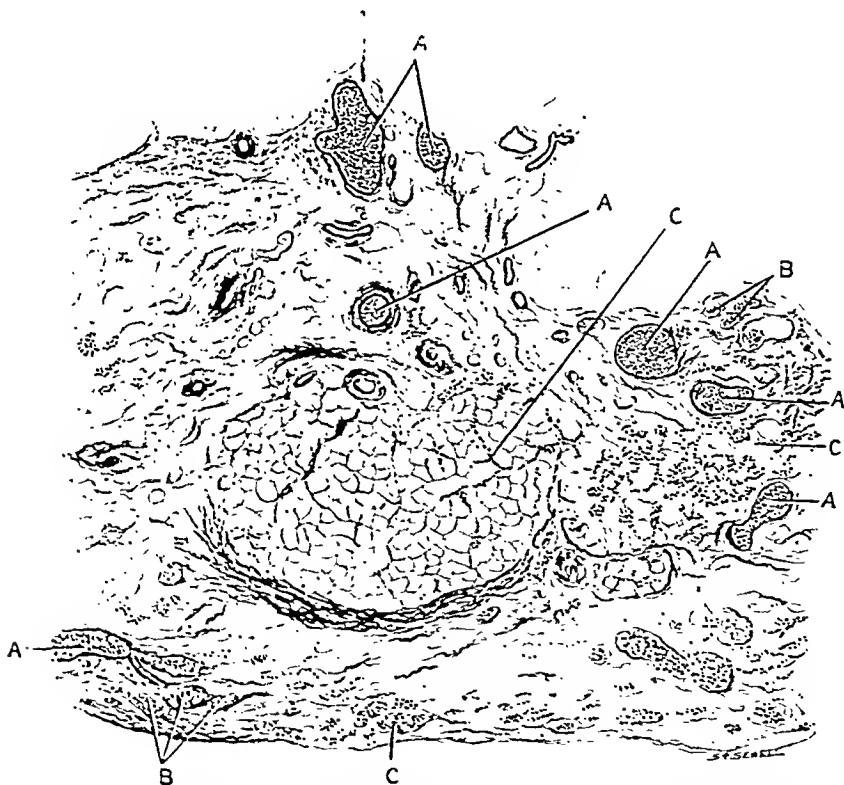


FIG. 351.—Drawing made from the tumour A of Fig. 350. A, Terminal ducts surrounded by their elastic coats. B, Acini. C, Epithelial-cell invasion of outside structures, including fat tissue. The dysgenetic epithelial hyperplasia in the ducts and acini is not papillary.

which a dysgenetic epithelial hyperplasia is strictly confined within ducts and acini. Such a change in the sequence of events is demonstrated by definite signs of malignancy as indicated by the character of the individual tumour

cells, which show nuclear hyperchromatosis, variation in size, and mitoses, although the process is still confined within ducts and acini (*State A, Class II*).

It is inevitable that difficulties should arise, when conjectures upon histological appearances have to be made without the aid of that biological evidence that could be attained only by experiment conducted upon human beings. The solution of these difficulties by this means is therefore impossible. Is it justifiable to fall back upon the evidence afforded by experiments conducted on animals? Whatever be the answer to this question, it is of extraordinary interest to study the animal experiments and compare their various stages with the changes that occur in the breasts of human beings. The time required to induce tar carcinoma in mice occupies a quarter or more of the animal's lifetime, during which period the skin to which the tar has been applied has gone through a sequence of pathological changes to which I have already drawn attention. An important point to realize is that in any one of these changes the process may be arrested, and the genesis of carcinoma may not mature. It is a fact that precisely the same pathological changes take place in the breast, and in the same sequence, viz., the desquamative hyperplasia, especially *Type B* in *Class I*, the dysgenetic hyperplasia of *Class II*, in which occur papillomata, and the conditions indicated in the *States A* and *B* of *Class II*. Lastly, when carcinoma eventually matures in human breasts, the total period of its induction usually corresponds to a quarter or more of a lifetime of the individual.

Can it be said that the states in the breasts of human beings correspond, in histological appearance and periods of their development, with those states seen in mice during the induction of tar cancer? Similarity of histological appearances cannot be denied; but there are those who deny, with some reason, that a quarter of a mouse's lifetime corresponds to a quarter of the lifetime in human beings. Fortunately, it is unnecessary to argue this point, because Harold Barnard, years ago, showed that the exposure of the human skin to tar occupied from twenty to thirty years before tar carcinoma ensued, and this evidence is available among workers in tar at the present time. He also pointed out that during this period a state of things appeared in the following sequence: warts; a state that microscopically looked like squamous epithelioma, but which was not accompanied by epithelial cell invasion or metastasis, and from which recovery could take place; and, finally, true squamous epithelioma with invasion and metastasis. Hence, because the knowledge concerning the lesions induced by the application of tar to mankind is well established, it is unnecessary to fall back on experiments on mice for the important evidence that connects the changing types of epithelial hyperplasia of the breast with those to be observed in the induction of tar carcinoma. I consider them to be identical in histological appearances, in the time occupied in their induction, in the similarity of their arrest in some instances, and in their slow or rapid progress in others. Hence I consider it wrong to regard the desquamative epithelial hyperplasia of *Class I* as being of no importance. I regard it as being a proemial state wherein an irritant remains in action undisturbed for a long time. In point of time it is too far away to be regarded as 'pre-cancerous'. There is at least one state that

occurs later in life than desquamative epithelial hyperplasia and is nearer to the carcinoma process (*State A, Class II*), and can with more reason be regarded as 'pre-cancerous'.

## *Class II.*—**DYSGENETIC EPITHELIAL HYPERPLASIA IN ACINI AND THE TERMINAL DUCTS WITH WHICH THEY COMMUNICATE.**

I do not intend to refer to purely duct carcinoma, which naturally belongs to this class. Neither shall I allude to duct papillomata, further than to point out that they occupy in sequence of breast events the same position

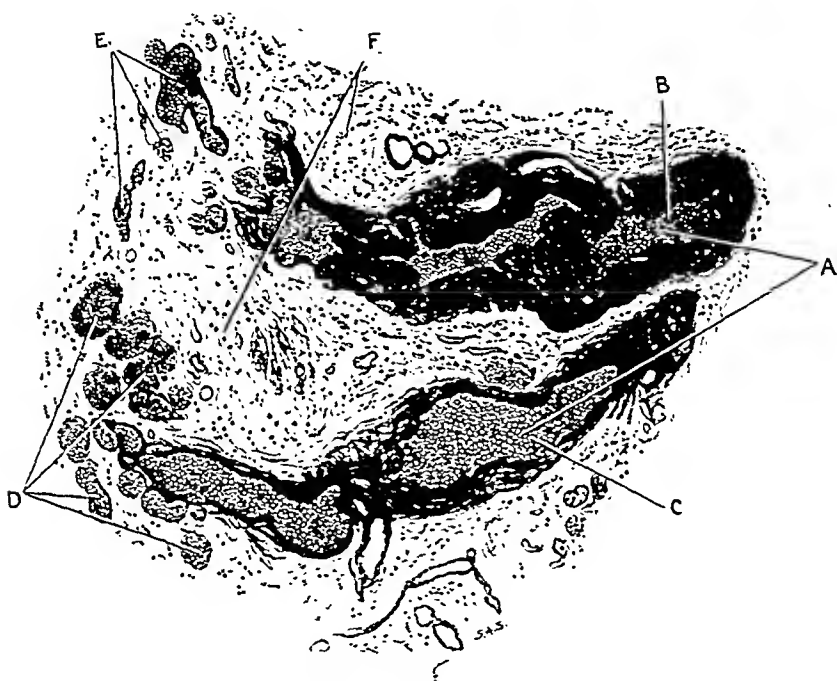


FIG. 352.—From the breast of a woman, age 38. Longitudinal section of terminal ducts, B, C, made just below bifurcation of a larger duct at A. D, E, Acini belonging to respective ducts, C and B. The elastica has been stained and has undergone enormous hyperplasia. There is marked epithelial cell invasion of tissues (F) outside the ducts and acini. The dysgenetic epithelial hyperplasia in duct and acini is not papillomatous. The tumour was not bigger than a finger-nail and occupied the outer part of the breast, between the nipple and the periphery of the gland. There was no affection of the lymphatic glands in the axilla. The Wassermann reaction was negative. (Mr. Percy Legg's patient.)

as that occupied by warts in the skin after the application of tar. I refer only to two kinds of dysgenetic epithelial hyperplasia (*States A and B*) that affect acini and the terminal ducts communicating with them. Before proceeding, it is essential that I should say that I do not consider that carcinoma of the breast of purely acinous origin has been demonstrated. In the earliest carcinoma in breasts (not purely of duct origin) I have never seen

a dysgenetic epithelial hyperplasia of an acinus that has not been accompanied by a similar process in the terminal ducts with which the affected acinus communicates. In many examples of early carcinoma the terminal duct implication of this kind is purely papillomatous in type, arising from the actual duct epithelium (*see Figs. 345-349*). This fact renders it impossible to infer that the duct is acting merely as a pathway by which the growth is spreading from the acinus. There is another fact that renders this inference impossible. It is this. I can show ducts (*Fig. 354*, for example) in which a



FIG. 353.—Longitudinal section of a terminal duct, A, A, and its acini, B, B, B, B. The elastica has been stained. The dysgenetic epithelial hyperplasia in only the main duct and one acinus has been drawn to show how full they are of growth (not papillomatous in type). The shaded ducts and acini were full of the same growth, which in all these structures was strictly confined within normal boundaries. This duct and its acini were discovered in the centre of an advanced carcinoma of the breast. At C, C there is epithelial-cell invasion of outside tissues. The woman was 49 years old, and died of the disease two and three-quarter years after the operation.

non-papillomatous dysgenetic epithelial hyperplasia is occurring that directly communicates with normal acini. The disease, in each instance, must be hyperplasia of duct epithelium as well as a hyperplasia of acinus epithelium, when the disease is present in the latter structure. One may have begun before the other, but as I have not seen a purely acinous affection in carcinoma of the breast, I cannot say in which structure the disease first began—although all my work indicates that it first appears in the duct.

Frequent examples of the histological appearances of *Class II*, which I am about to describe, are to be observed in breasts where there is early

Paget's disease of the nipple. This disease, when it affects the nipple, is commonly complicated by the development of primary carcinoma in the ducts or terminal ducts and acini in the breast. Hence one would expect to see either dysgenetic forms of epithelial hyperplasia immediately antecedent to carcinoma (*State A*), or early carcinoma itself (*State B*). It is essential in this investigation that all specimens prepared for microscopical examination should be stained to show the elastica. In the early stages of dysgenetic epithelial hyperplasia the elastica has not been destroyed—by distention or otherwise—and its presence is invaluable in enabling the observer to distinguish between ducts and acini and between ducts and lymphatic vessels and spaces.

I divide *Class II* into two states: *State A*, in which the dysgenetic epithelial cells are strictly confined within terminal ducts and acini; and

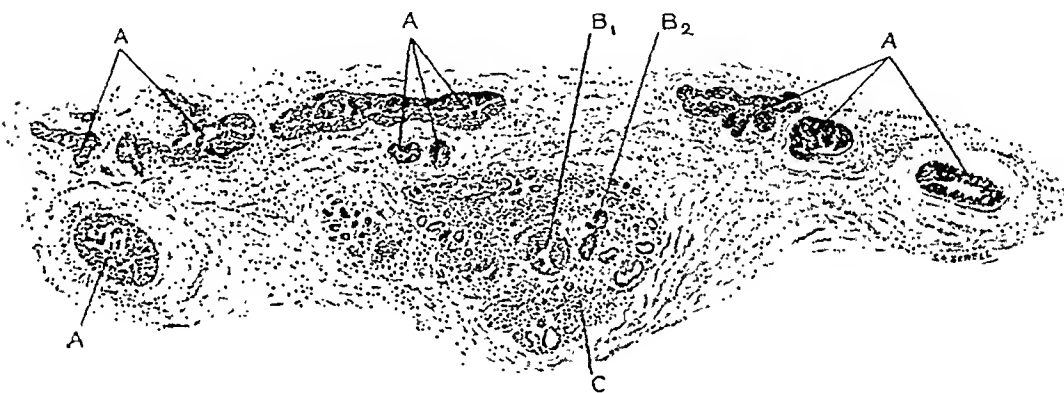


FIG. 354.—A, Longitudinal section of a convoluted duct. C, A lobule in which branches of the duct terminate at B<sub>1</sub> and B<sub>2</sub>. The elastica is not stained in this section, but it was in other sections, and had undergone enormous hyperplasia throughout the whole breast. The figure is part of a whole section of the gland. The ducts show a dysgenetic epithelial hyperplasia, not papillomatous, and strictly confined within normal boundaries. There was no epithelial growth in any acini. The woman was 38 years old. There is epithelial cell invasion near this part of the gland, and no other focus of dysgenetic epithelial hyperplasia could be found in the breast than that which exists in this duct. The lymphatic glands in the axilla did not contain carcinoma. The Wassermann reaction was negative.

*State B*, in which the dysgenetic epithelial cells have at some part, or parts, escaped from the affected terminal ducts and acini, and have appeared in the surrounding connective tissue. Metastasis may have also occurred, and a state of carcinoma have been established.

*State A*.—Figs. 328–340 and 355 represent the histological appearances of *State A*, and are taken from seven different patients. In all of them only terminal ducts and acini are the parts affected by dysgenetic epithelial hyperplasia. Figs. 328 and 336 are the biggest lesions I have of this state. In other specimens, where the same tumour formations in the ducts and acini have been larger than this, they have always been carcinomatous (see Figs. 346 and 349) and belong to *State B*. The lesions in *State A* usually occur in younger women than the early carcinomatous lesions described in *State B*.

The significance of these two facts may be that *State A* is a state from which carcinomata (*State B*) may emerge.

The acini in *State A* are all of the same histological appearance. They are filled or partially filled by sessile dysgenetic epithelial growth, and only occasionally is there a suggestion of papillomatous formation. The ducts, however, do not always present the same histological appearances. In *Figs. 328-336* the dysgenetic epithelial growths in the ducts are papillomatous, and in those of *Figs. 340* and *355* the growths of epithelial cells contain no fibrous-tissue elements.

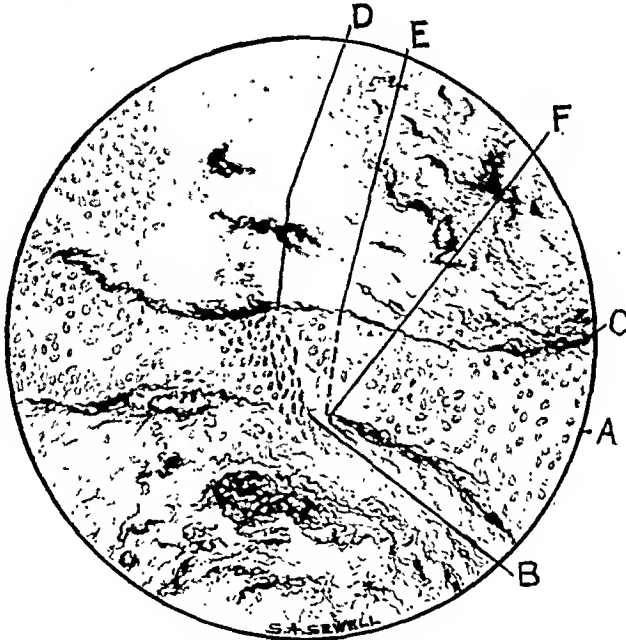


FIG. 355.—A, Longitudinal section of terminal duct. C, Elastica, stained. At B epithelial cells are emerging from the interior of the duct into the outside tissues. The dysgenetic epithelial hyperplasia in this duct is not papillomatous in type. There is enormous hyperplasia elastica in other parts of this breast, and the Wasserman reaction was positive. D, E, F, see text, p. 530. From a woman, age 42.

In the vast majority of the affected ducts and acini in these breasts in *Class II, State A*, there is not the slightest suspicion on microscopic examination, of epithelial cells that have transgressed their normal boundaries. Yet I regard *State A* as being second in importance only to carcinoma itself. Consequently it may well be asked, if I consider that to be so, why is not an intermediate stage between *States A* and *B* more demonstrable than in any specimen I have shown. In reply I would draw attention to *Fig. 333*, reproduced from *Fig. 330*, where there is a state of things that can be explained by an epithelial cell invasion; but as these histological appearances are open to other explanations, I do not reproduce them as an example of epithelial invasion. Further, the examples I have given in *State B* are such early stages of carcinoma that I consider they may be regarded as being almost within the realm of a transitional stage. *Figs. 327, 329, and 332*

illustrate the connection there is between *Type B* of *Class I* and *State A* of *Class II*. Colostrum-like cells are still present in these specimens. It might be said that these colostrum-like cells are a result of desquamation from the surface of the tumour, but there are no signs of their being formed from the cells at this part of the tumour, and they can be seen developing from otherwise normal cells of the duct wall. However, it is a fact that colostrum-like cells are sometimes thrown off by desquamation in all kinds of dysgenetic epithelial hyperplasia in ducts (see *Fig. 328*), but this fact may emphasize rather than disconnect the alliance that appears to exist between *Type B*, *Class I*, and *States A* and *B*, *Class II*. In describing *State A*, I wish particularly to analyse the appearances seen in *Fig. 336*. Very few seem able to realize, without its being pointed out, that *A*, *A2*, *B1*, *B2*, *B3*, and *B4* are all terminal ducts containing papillomatous and sessile dysgenetic epithelial hyperplasia, and that they directly communicate with separate clusters of acini, which are distended by dysgenetic epithelial hyperplasia. In fact the terminal ducts and acini of one main duct alone are the only parts affected. The same appearances are seen in *Figs. 328* and *330*, which are cut from another angle. In many parts of *Fig. 336* the acini, distended with growth, are becoming confluent. What could be the result of progress in the epithelial hyperplasia of *Fig. 336*? It might be said that the tumour may grow to an enormous size and be composed of cysts filled by epithelial cells and all contained within normal but grossly distended structures. An appearance such as this has never been seen, except in the presence of carcinoma (see *Figs. 346* and *349*). Hence I believe that the only result of progression in *State A* must be carcinoma; a remarkable example of this process, as a progression from *State A* to *State B*, is that depicted in *Fig. 346*, where half the breast is occupied by a large carcinoma. The size of this tumour is mainly due to the dysgenetic epithelial hyperplasia that still occupies distended but otherwise normal boundaries (see *Figs. 347* and *348*, also compare with *Figs. 349* and *353*), and the increase of size of this tumour that can be allotted to infiltrating epithelial cells is remarkably small. The patient from whom this picture was photographed died of the disease two years after the operation. There can be no doubt that *Fig. 336* and *Fig. 316* form a remarkably connected pair.

Now, to call more particular attention to the sections of whole breasts reproduced in *Figs. 328*, *330*, and *336*. In these specimens, very interesting and diffuse dysgenetic epithelial hyperplasia can be seen. The growths are occurring in pre-existing cysts. Nobody can doubt that the terminal ducts and acini were dilated before the growth occurred. There are so many empty cysts in the neighbourhood of the growths, and the dilatation of the respective ducts at the nipples in *Figs. 328* and *330* are so separated from the dilated terminal ducts and acini into which they directly lead, that it is impossible to assume that these dilatations near the nipple could have occurred as secondary processes. In the terminal ducts of the three specimens, the growths are a mixture of papillomatous and sessile dysgenetic epithelial hyperplasia. The remarkable fact which concerns these three specimens is that in each the terminal branches of only one main duct have been affected—a fact that has been demonstrated by cutting whole

sections, in series, of these breasts. The importance of the fact lies in the obvious inference that only one duct contained the irritation necessary to induce the hyperplasia. Although the growths are strictly confined, in most parts, within terminal ducts and acini, they give rise to very suspicious histological appearances.

In connection with the restriction which dysgenetic epithelial hyperplasia in *State A* shows to normal boundaries, I must refer to an important duct in *Fig. 354*. In it there is a dysgenetic epithelial hyperplasia, sessile (not papillomatous) in type. The peripheral half of this duct is filled and distended by living epithelial cells that have grown from its walls. I could not discover any acinous affection (*see* lobule C, *Fig. 354*). Serial sections of the whole of this breast did not reveal any other form of dysgenetic epithelial hyperplasia. Although I could not discover in any part of this duct a place from which epithelial cells were escaping, yet from this duct epithelial cells must have escaped to create the early state of carcinoma that was present in this breast. It is difficult to know whether to allot this duct to *State A* or *State B*. This difficulty of allotment emphasizes the importance of *State A*. Compare this specimen with the longitudinal section of the terminal duct from another breast, *Fig. 355*. At one point (B) epithelial cells are pouring out of the duct into the surrounding tissue, and the presence of carcinoma is certainly established. Now, suppose this duct had been cut and viewed transversely at the dotted line between E and F, i.e., in immediate juxtaposition to, but not including, the part where epithelial cell invasion is occurring, all that would have been visible in this imaginary transverse section would have been the distention of the duct by a dysgenetic epithelial hyperplasia; but, as there would have been no indication of cells passing through its walls, nobody would be justified in assuming it to be malignant; and yet it must be part of the carcinoma process, and a most intimate part, for exactly similar cells are in immediate contact with it invading the surrounding tissue. Further, what could be said of the epithelial cells at D? They must surely be malignant, or potentially malignant. I must leave it to the imagination and point of view adopted by my readers to make their own decisions on this matter.

I consider the dysgenetic epithelial hyperplasia, at the point of my imaginary transverse section, although the cells are strictly confined within the duct walls, to be malignant. I make the same conclusion concerning the epithelial cells contained in the affected ducts and acini in the carcinomata from which *Figs. 345, 347-353* are taken. I believe that *State A* of *Class II* is a potentially malignant state, and that, should progress continue, the catastrophe of carcinoma is only a question of time. Whatever my belief is in this matter, there can be no doubt that *State A* is histologically more nearly connected with *State B* than any other pathological process in the breast; but no definite statement can be made as to whether it is only a fire prepared for the match which lights the conflagration of carcinoma, or whether it is a definite preliminary stage of carcinoma. Hence, if there must be the term 'pre-cancerous', I would apply it to this state. In making this statement, I would lay down as a law that there is no kind of epithelial hyperplasia that will inevitably end in carcinoma.



*State B.*—Here the dysgenetic epithelial hyperplasia is limited to acini and terminal ducts, and has precisely the same histological appearances as those in *State A*. Epithelial cells have invaded the surrounding tissues and may have undergone metastasis.

There is no doubt whatever that carcinoma exists in all the tumours I describe in *State B*. Figs. 345–355 are taken from early or advanced carcinomata removed from eight different breasts. In all of them the acini and terminal ducts are the only parts affected (except in the advanced carcinoma of Fig. 346, where two ducts as well as their acini are completely filled with dysgenetic epithelial hyperplasia from the ampulla downwards). The dysgenetic epithelial hyperplasia in the acini and terminal ducts are the same in histological appearances as those in *State A*. Sometimes the duct tumours are papillomatous, and sometimes purely sessile and non-papillomatous (*see Figs. 351–355*). As growth of the tumours proceeds, the intra-duct and intra-acinus tumours become more rampant (*see Figs. 347, 348, 349, and 353*, which were taken from three advanced carcinomata). Curiously enough, the tumours in the ducts and acini, although becoming rampant, still maintain their papillomatous or non-papillomatous state until all traces of duct and acinus walls are completely destroyed. The maintenance of papillomatous or non-papillomatous states respectively throughout the lives of these tumours indicates some method in their madness.

From some part of the dysgenetic epithelial hyperplasia epithelial cells have escaped, and they have invaded surrounding tissues and metastasis has occurred. It is difficult to detect the exact regions of ducts and acini from which the escapes of epithelial cells were made, even from an examination of whole sections cut in series. Fig. 355 was a lucky discovery.

### SUMMARY AND CONCLUSION.

1. There appears to me to be a definite sequence of events in epithelial changes in the breast that end in carcinoma. In making this statement I do not wish it to be inferred that every event in the sequence invariably occurs—for instance, papilloma may be absent, and its place may be represented by a sessile and non-papillomatous state. One or more of these stages may be absent in the development of acute carcinoma.

2. It is most significant that the different epithelial changes that I have described have a common site of origin, namely, the acini and terminal ducts.

3. It is important to realize that the chain of events may be interrupted at any point, due either to intrinsic biological changes in the cells or to extrinsic factors such as variation in connection with the exciting cause. The interruption may result in the process remaining latent or in its subsequent degeneration and disappearance.

4. The sequence of events to which I have called attention begins as what I have termed a desquamative epithelial hyperplasia, and passes on to an epithelial hyperplasia which is not desquamative and which I have termed dysgenetic epithelial hyperplasia. Included in the latter, and following desquamative epithelial hyperplasia, are epithelial changes which occur in the following sequence:—

*a.* A condition in which the epithelial cells retain their normal structure and produce papillomata confined within the acini and terminal ducts.

*b.* An epithelial hyperplasia in which the individual cells are showing signs of beginning malignancy—e.g., nuclear hyperchromatosis, variation in size of the cells, and mitoses—yet the process is still confined within acini and terminal ducts and has not extended into the surrounding connective tissue (*State A, Class II*). This is the state which immediately precedes carcinoma, and hence is the 'precancerous state'.

*c.* A more advanced condition in which, in addition to the cell changes, the process has extended beyond the walls of the acini and terminal ducts and has invaded the surrounding connective tissue (*State B, Class II*).

5. The fact that one or more of the various epithelial changes, particularly the 'precancerous state', is found in the centre of fully developed carcinoma is most significant.

6. The events I have described as occurring in the breast are identical in time and sequence with those occurring in the skins of man and mice after the application of tar.

It is a pleasant duty to thank Dr. J. A. Murray, the St. Bartholomew's Hospital Surgical Clinic, Mr. Percy Legg, and Dr. W. R. Smith for the valuable and interesting material they have placed in my hands. At the same time, it is only fair to these gentlemen to state that they are not responsible for the conclusions I have reached. Finally, I must thank Mr. F. F. Burghard and Dr. May Cutler for making clear parts of this article which appeared obscure.

**FURTHER RESULTS OF NERVE ANASTOMOSIS.\***

AN ILLUSTRATED RECORD OF SOME EXPERIMENTS IN WHICH:—

1. THE CENTRAL AND PERIPHERAL ENDS OF A DIVIDED NERVE WERE IMPLANTED AT VARYING DISTANCES APART INTO A NEIGHBOURING NORMAL NERVE.
2. CERTAIN NERVE-TRUNKS OF THE LIMBS WERE DIVIDED AND ANASTOMOSED BY SUTURE IN CROSS-WISE FASHION.

By SIR CHARLES BALLANCE,  
LIONEL COLLEDGE, AND LIONEL BAILEY, LONDON.

THE finest sutures and needles have been employed in all the experiments. Sutures used have been Van Horn arterial silk, and linen thread and catgut of about the same size. It does not appear that there is, on examination of microscopical sections, any advantage gained in preferring one kind of material for suture to another. All sutures are dead foreign material, and as such are absorbed. The initial cellular proliferation which ultimately encapsulates and absorbs the suture appears to be much the same in amount whatever material is employed, provided the suture is of the same size; the only difference consists in the time taken in the process of substitution and absorption, i.e., the time required for the replacement of the suture material by living tissue to be complete.

An attempt has been made in each experimental anastomosis to pass the needle and thread only through the neurilemma, but the nerves experimented with are so minute that this attempt must have failed in many instances. The results obtained in these experiments seem to indicate that the passage of the needle and suture only through the neurilemma, though theoretically desirable, is not essential to success in nerve suture.

It has sometimes been said that, in suturing a large nerve-trunk of a limb end to end, corresponding nerve-fibres should be brought into contact. This is impossible in any case: the suggestion is theoretical and is of no practical importance. But on looking at the transverse section of a nerve-trunk it is seen to be made up of bundles of nerve-fibres, some larger than others. The anatomical arrangement of nerve bundles does make it possible in some cases when suturing large nerves end to end to bring corresponding nerve bundles in apposition; but this is impracticable when the minute nerves of the neck of the monkey are the subject of experiment.

Sir David Ferrier, who has taken much interest in these experiments, was anxious that nerve-ends should be brought together and fixed in position without the aid of sutures. For this purpose various varnishes were tried,

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\* See "Some Results of Nerve Anastomosis," *BRITISH JOURNAL OF SURGERY*, Vol. xi, p. 327. The Medical Research Council defrayed the expenses of this research, and gave me every assistance in the performance of the experiments, which were carried out at the National Research Institute, Hampstead.—C. B.

such as sandarach varnish, benzo-mastiche varnish, and acetone collodion. It is obvious that no particle of a drop of these varnishes could be allowed to come in contact with the cut end of a nerve, and their use has been given up. The discovery of a living muscle varnish has, however, been successful. The varnish is made by cutting living muscle into small pieces, adding a few drops of sterile salt solution, and pounding the mixture in a sterile mortar with a pestle till a sticky mass is formed. Presumably the sarcolemma is ruptured and the sarcons material within is set free.

In double lateral implantation of one nerve into another, the time required for functional recovery varies with the distance apart of the implanted ends of the nerve, and possibly also with the method of implantation employed. The greatest distance apart of the ends of the external popliteal nerve when implanted into the internal popliteal nerve has been in these experiments 5 cm. But the distance apart of the sites of implantation of one end of the musculospiral nerve into the median nerve at the lower margin of the axilla, and of the other end of the musculospiral nerve into the median nerve just above the bend of the elbow, is much greater than 5 cm. In a small rhesus monkey in which this experiment (not yet complete) was carried out, the distance apart of the two ends of the musculospiral nerve when implanted into the median nerve was fully  $8\frac{1}{2}$  cm. ( $3\frac{3}{8}$  in.). Fifteen months after the experiment was performed all the muscles of the forearm contracted to a medium faradic current.

In man, massage and electricity are available to maintain the nutrition of the muscles, and the limb is splinted to prevent lengthening of tendons and muscles during the period required for nerve regeneration; but in monkeys none of these measures is possible. The ankle- and wrist-joints tend to become flexed, and the toes and fingers to curl up, so that it is not true to speak of the recovery that does take place as perfect or complete. In dogs, on the other hand, in which double lateral implantation of the external popliteal nerve into the internal popliteal nerve has been done, the shortening of the flexor muscles and tendons does not take place, as in the act of walking the toes and ankle are by the natural movements extended. In a greyhound, after ten months had elapsed from the date of operation, in which the two ends of the external popliteal nerve were attached to the internal popliteal nerve 5 cm. apart, a median faradic current produced strong contraction in the tibialis anticus, with extension of the ankle, but the toes were only weakly extended. The greyhound could gallop fast over the ground at this date without any apparent disability. The extension of the toes in the dog is normally a weak movement, and the greyhound in galloping and jumping is dependent chiefly on the strong action of the flexors of the ankles and knees of the hind limb. The plan adopted of attaching the ends of the external popliteal to the internal popliteal nerve in this experiment is shown in *Fig. 356, c*.

When an opportunity offered for stimulating directly the nerves in double lateral implantation after functional recovery and before the death of the animal, a notable result was obtained. On faradic stimulation of the peripheral segments of the two nerves it was found that a stronger current was required to produce the same effect than was necessary when faradic

stimulation was applied to the central segments of the two nerves—in other words, the same strength of current caused less and less response as the stimulation proceeded from the central to the peripheral parts of the nerves.

This phenomenon has been described as the phenomenon of the avalanche (see G. Durante's article in Cornil and Ranvier's *Manuel d'Histologie pathologique*, 3rd edition, 1907, Vol. III, p. 495: "Phénomène de l'Avalanche (Pflüger), de la Boule de Neige (Chauveau)"). Durante points out, on p. 603, that functional restoration may occur ("transmission active de neuroblaste à neuroblaste: conductibilité plasmique") while there is still incomplete regeneration of the nerve-fibre, and that it is important to distinguish this function of conductivity from that of excitability. He describes the latter as follows: "pouvoir de transformer des vibrations (excitations) diverses en des vibrations nerveuses". There is in the above experiment a clear differentiation between the phenomenon of conductivity and that of excitability. The difference in functional activity of the nerve-fibres, in the central and peripheral segments of the nerves, appears to depend on a difference in histological or anatomical structure—in other words, on the greater or less perfection of the process of regeneration attained by them.

It remains to discuss the best method of attaching the two ends of a divided nerve in double lateral implantation to a neighbouring nerve (*Fig. 356*). Is it better to make transverse incisions, at a certain distance apart, in the course of the nerve into which the two ends of the implanted nerve are to be sutured; or is it better to implant the two ends into median incisions? At this stage of the experiments it does not seem possible to come to any conclusion. It would seem, however, that when median incisions are made in the receiving nerve, there is a tendency, in the case of double lateral implantation of the external popliteal

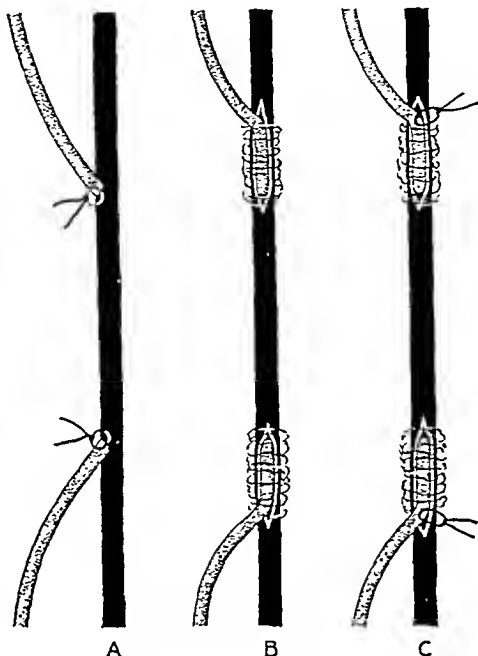


FIG. 356.—Diagrams of the methods employed in double lateral implantation for fixing the ends of a divided nerve to a neighbouring nerve. A, Two transverse incisions were made in the side of the normal nerve for about one-third of its diameter. The ends of the divided nerve were fixed into the incisions by one suture of iron-dyed silk, linen thread, or catgut. The sutures were the finest obtainable (arterial size) and were threaded on the finest needles. The attempt was always made to suture sheath to sheath, but it is not claimed that this was always accomplished. B, Implantation of the ends of the divided nerve into median incisions made in the normal nerve. Fixation was effected by 'muscle-varnish' without sutures. C, Implantation of the ends of the divided nerve into median incisions made in the normal nerve. Fixation was effected by 'muscle-varnish' and by one suture; each suture passed through the sheaths of both nerves half an inch from the end of the divided nerve.

nerve into the internal popliteal nerve, for the two ends to become connected with fibres originally belonging to both the external and the internal popliteal nerves, so that on proximal stimulation of either nerve the response may be, not a pure flexor or extensor one, but mixed flexor and extensor, one movement, however, being more marked than the other. When incisions are made in the side of the normal nerve, a portion of it is cut off from its previous connections both distally and proximally. This isolated band of nerve-fibres, together with the connective tissue of the sheath, is the site of a cellular proliferation. There is thus formed a new tissue through which nerve-fibres penetrate or develop, and ultimately produce a functional union between the two ends of the divided nerve. In such an experiment, on faradic stimulation of the proximal segments of the two nerves, pure effects of flexion and extension of ankle and toes have been observed. But this is probably not true of all such experiments, for on examination of some of the microscopical drawings the nerve-fibres of the central and peripheral ends of the external popliteal nerve are clearly connected with nerve-fibres belonging to both internal and external popliteal nerves. Nerve-fibres have a spiral arrangement in a nerve-trunk, and hence what appears to be clearly exhibited in a microscopical drawing may not indicate the true functional connections of the nerve-fibres.

In the notes of the experiments, the dates on which faradic stimulation was applied to the previously paralysed muscles are given. It is desirable to remember that this date, if a response to the electrical stimulation occurs, is not the date of the recovery of the muscle. The frequent etherization of a monkey for the purpose of determining the earliest date at which a response follows faradic stimulation is impossible. The risk to the animal, and possible failure of the experiment, which in any event requires that the monkey should live for many months, makes it essential that an anæsthetic should only occasionally be administered.

The microscopical sections have been cut, stained, and mounted by Mr. Steward at the Royal College of Surgeons, by the kind permission of Sir Arthur Keith.

### DETAILS OF EXPERIMENTS.

*Experiment 1.*—Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.

**RHESUS MONKEY.**—External popliteal nerve divided and attached in two places to incisions made in the side of the internal popliteal nerve  $\frac{1}{2}$  in. apart; one suture used for each anastomosis, of iron-dyed silk (Van Horn). Operation on Dec. 5, 1922.

*Jan. 17, 1923.*—Foot-drop marked.

*April 26.*—Climbs well with both feet.

*May 23.*—*Faradic stimulation.* Definite contraction (slow response) in tibialis anticus and peronei muscles; none in extensor longus digitorum or extensor hallucis. Flexors of ankle contract almost normally.

*July 19.*—*Faradic stimulation (6½ months).* Extensor communis digitorum contracts. It is not possible in the monkey to keep the ankle and toes extended during the period of nerve regeneration, hence the toes are somewhat curled up in walking.

Oct. 23.—*Faradic stimulation.* All the muscles of the foot and toes supplied by the external popliteal nerve contract, but they all require a somewhat stronger current than the corresponding muscles of the other or normal limb. Galvanic response normal.

Nov. 20.—11½ months. The animal was ill, and hence was killed by ether inhalation. Before death occurred the anastomoses were exposed and stimulated with the faradic current. (Sir David Ferrier was present.)

*Results of Stimulation (Fig. 357):—*

- B and D, Flexion of ankle and toes.
- A and C, Extension of ankle and toes.
- E, Extension of ankle and toes.
- F, Flexion of ankle and toes.

*Microscopical Appearances.*—The proximal segment of the external popliteal nerve ends in a bulb (Fig. 358), and from it nerve sheaths extend downwards, apparently in new tissue on the outer aspect of the internal popliteal nerve-trunk between the two anastomoses.

The distal segment of the external popliteal nerve (Fig. 359) exhibits complete regeneration, its nerve-fibres being in direct communication with those of the proximal segment in the tissue on the outer side of the internal popliteal nerve-trunk. The activity of regeneration appears to be not less in the lower segment than in the upper segment.

Proximal to the bulb the main nerve-trunk stains deeply, as does also the whole length of the uninjured internal popliteal nerve. In the bulb the sheaths are finer, very numerous, and lightly stained. They interlace in many directions, but mainly tend to run longitudinally. At the level of the bulb there is a place on the outer side of the internal popliteal nerve where regeneration has taken place, and here also the neurilemma sheaths are fine, numerous, and not deeply stained. The same is true of the region of the distal anastomoses. The sheaths stain lightly, they interlace, and are numerous. Beyond the anastomosis area the sheaths of the main trunk of the regenerated distal segment of the external popliteal nerve stain deeply. The new tissue between the proximal and distal segments of the external popliteal nerve is permeated by numerous groups of stained sheaths running in a longitudinal direction. They do not seem to have any connection with the internal popliteal nerve. Wherever regeneration has taken place are seen numerous neurilemma cells with rod-shaped nuclei. These lie alongside the new sheaths. Connective-tissue cells with oval nuclei are present also.

Before the death of the monkey the upper part of the opposite Rolandic area was stimulated. Extension of the ankle and toes occurred at a spot a little above the area where stimulation caused flexion of the ankle and toes. These results of stimulation of the cortex were like those obtained by stimulating the corresponding areas of the opposite Rolandic cortex.

*Experiment 2.—Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.*

Cat.—External popliteal nerve divided and the ends implanted in two places into the internal popliteal nerve, 4 cm. apart. The ends of the external popliteal nerve were inserted into longitudinal slits made in the middle of the internal popliteal nerve. The anastomoses were fixed in position with 'muscle varnish'. An inch from each anastomosis two drops of 'benzo-mastiche' varnish were applied to each segment of the external popliteal nerve. This varnish fixed the proximal and distal parts of the external popliteal nerve to neighbouring muscular tissue. It was hoped in this way to secure the anastomoses from displacement, as no sutures were used.

2 months and 20 days. The animal suffered from diarrhoea and was killed by ether inhalation.

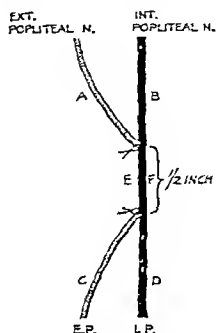


FIG. 357.—*Experiment 1.* Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 11½ months.

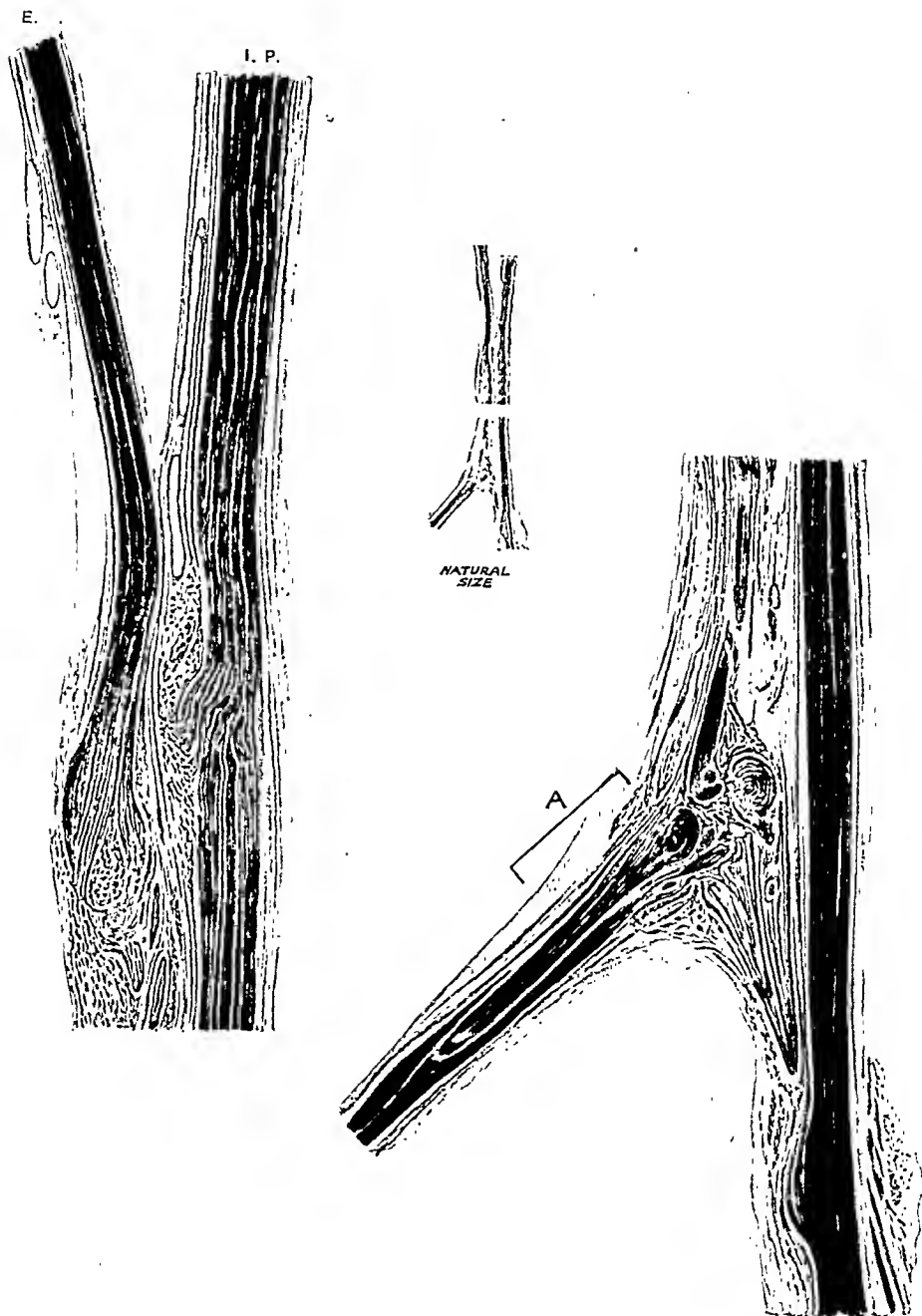


FIG. 358.—*Experiment 1. Monkey.* External popliteal nerve divided and the ends anastomosed in two places, half an inch apart, to the side of the internal popliteal nerve. Specimen obtained  $11\frac{1}{2}$  months afterwards. Weigert stain. I. P., Internal popliteal nerve; E. P., External popliteal nerve; A, Portion shown more highly magnified in Fig. 359. (*N.T.*)



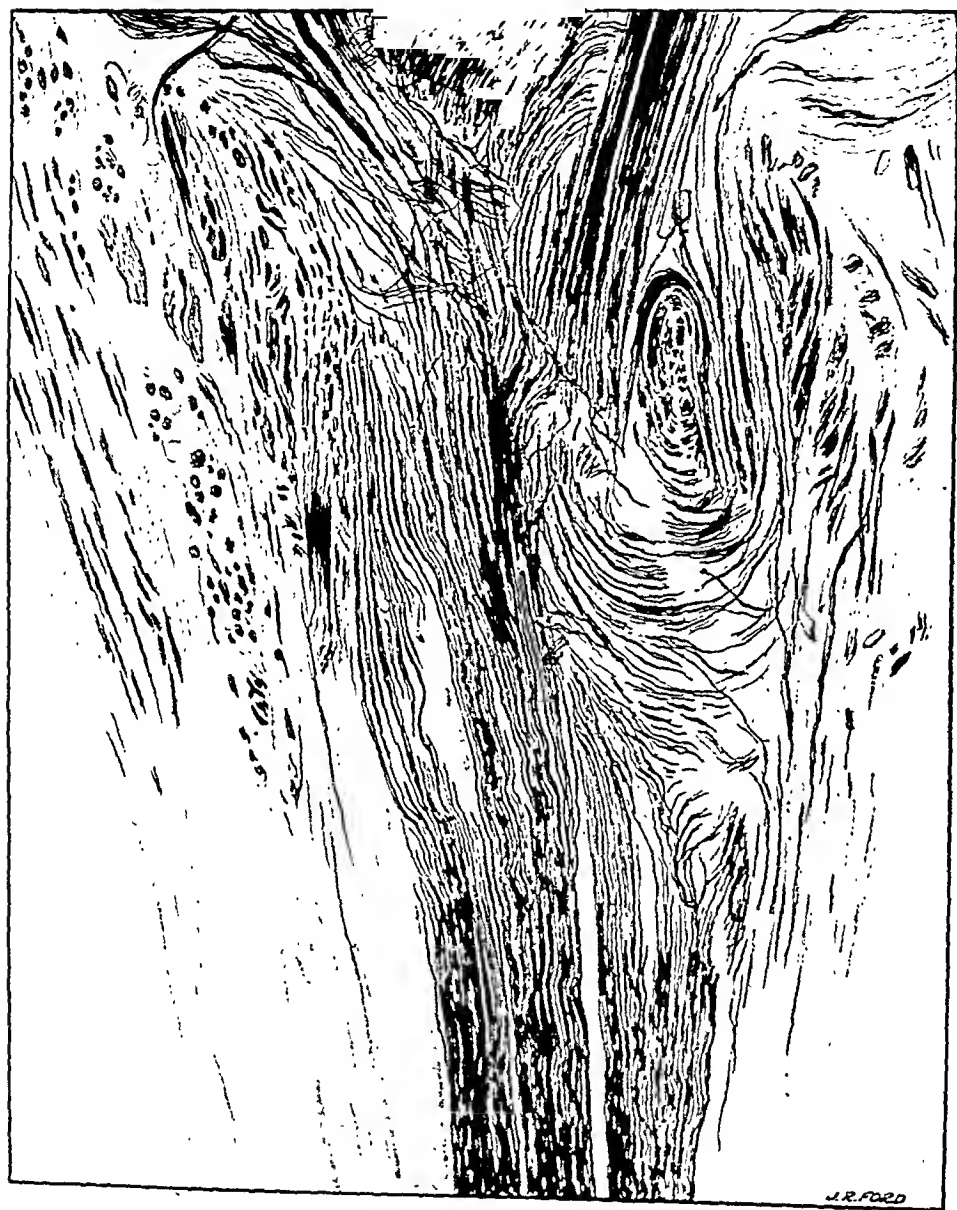


FIG. 359.—Distal part of external popliteal nerve marked A in Fig. 358. ( $\times 42$ .)

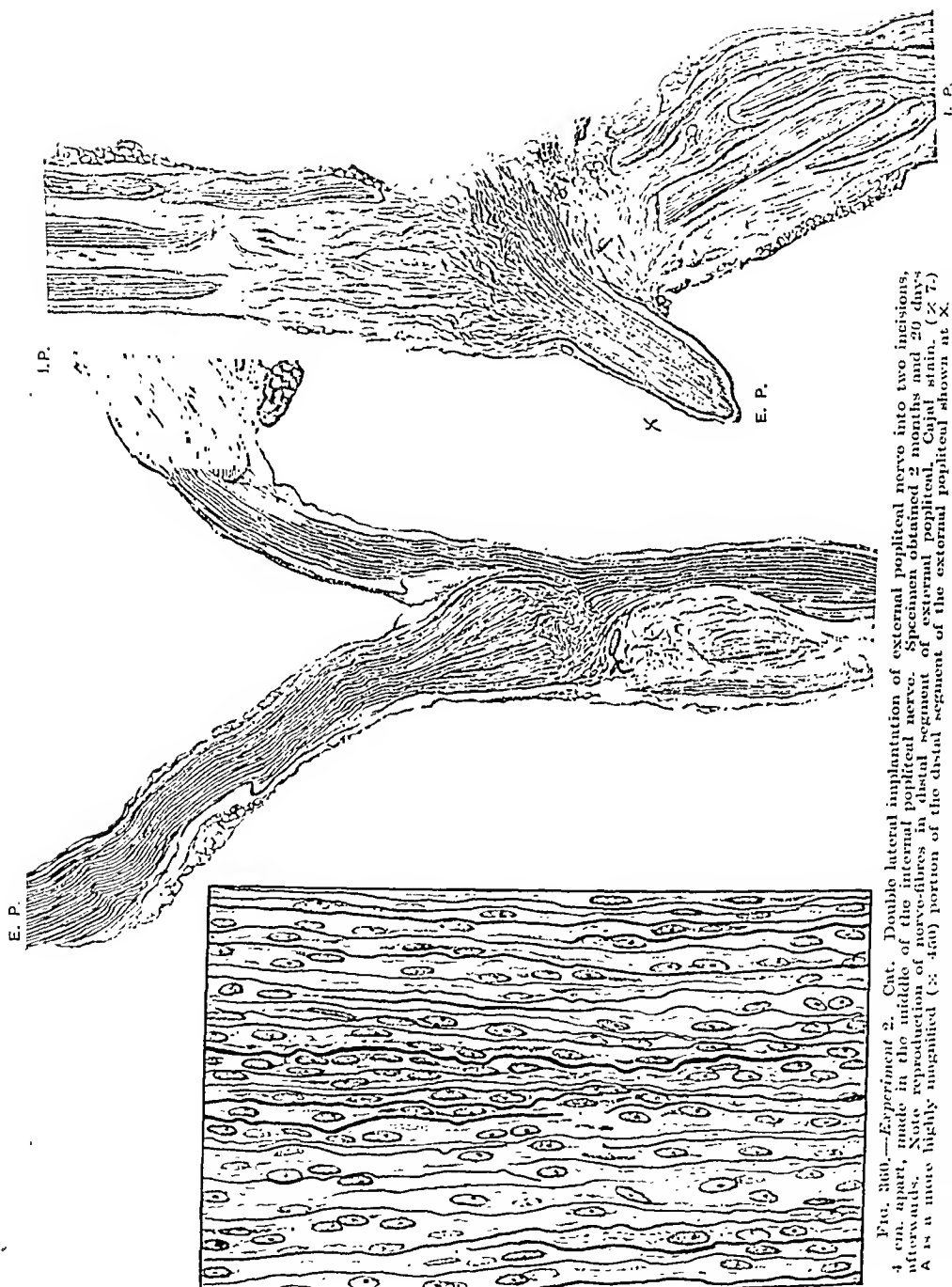


FIG. 360.—Experiment 2. Cut. Double lateral implantation of external popliteal nerve into two incisions, 4 cm. apart, made in the middle of the internal popliteal nerve. Specimen obtained 2 months and 20 days afterwards. Note repopulation of nerve-fibres in distal segment of external popliteal. Cujal stain. ( $\times 7$ ). A is a more highly magnified ( $\times 400$ ) portion of the distal segment of the external popliteal shown at X.

*Microscopical Appearances.*—In the low-power drawing (*Fig. 360*) it is seen that the distal end of the proximal part of the external popliteal nerve ends in a bulb, some of the fibres of which join fibres of the internal popliteal nerve, while others pass into the connective tissue on the outer side, in which are other columns of nerve-fibres.

The distal segment of the external popliteal shows marked regeneration of nerve-fibres. The proximal end of the distal segment spreads out in a wide bush of fibres which spread upwards to join nerve columns from the outer to the inner border of the internal popliteal nerve. These newly regenerated fibres are finer than those which are seen in undamaged nerve-trunks (*Fig. 360. A*).

It would appear from the position of the two ends of the external popliteal nerve that both had shifted after the operation: the ends of the proximal segment upwards from its position in the proximal median incision in the internal popliteal nerve, and the end of the distal segment downwards from its original position in the distal median incision in the internal popliteal nerve.

*Experiment 3.*—Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.

**RUEST'S MONKEY.**—External popliteal nerve divided and  $\frac{3}{4}$  in. of the nerve cut away. Ends of external popliteal nerve implanted into incisions made in the side of the internal popliteal nerve, 1 in. apart. Two sutures of iron-dyed arterial silk employed to fix each anastomosis.

$\frac{1}{4}$  months.—No response to faradic current in extensors of ankle and toes.

$\frac{7}{10}$  months.—Response to stimulation with faradic current: Tibialis anticus and extensor communis digitorum. (Sir David Ferrier present.)

$10\frac{1}{2}$  months.—Toes still curled up. Ankle cannot be extended beyond a right angle because of shortening of flexor tendons and muscles. Animal was ill, hence was killed by ether inhalation.

Before death the muscles were stimulated with the faradic current through the skin: *All the extensor muscles of the ankle and toes responded to a medium current, also the peronei muscles: the flexors also all contracted.*

The anastomoses and nerves were exposed and stimulated. A weak faradic current was used.

*Results of Stimulation (Fig. 361):—*

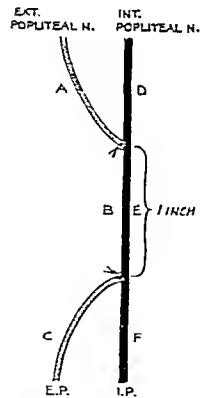
A, B, and C, Contraction of all the extensors and the peronei.

D, E, and F, Contraction of all flexors of ankles and toes.

On stimulating A and D the strongest movement was exhibited.

„ „ C and F the movement was markedly less.

„ „ B and E the weakest movement of either flexion or extension was observed.



**FIG. 361.**—*Experiment 3.* Diagram showing the points on the popliteal nerves at which faradic stimulation was applied.  $10\frac{1}{2}$  months.

*Microscopical Appearances.*—We have no record of the distal anastomosis as this was unfortunately lost.

The proximal anastomosis is shown in *Fig. 362*. The site of the incision in the internal popliteal nerve is obvious: it exhibits a plexus of nerve-fibres spreading in many directions.

Below the anastomosis the external popliteal nerve is continued in the new tissue formed external to the uninjured part of the internal popliteal nerve, though some fibres appear to join those of the internal popliteal nerve at the site of the anastomosis.

E. P.

I. P.

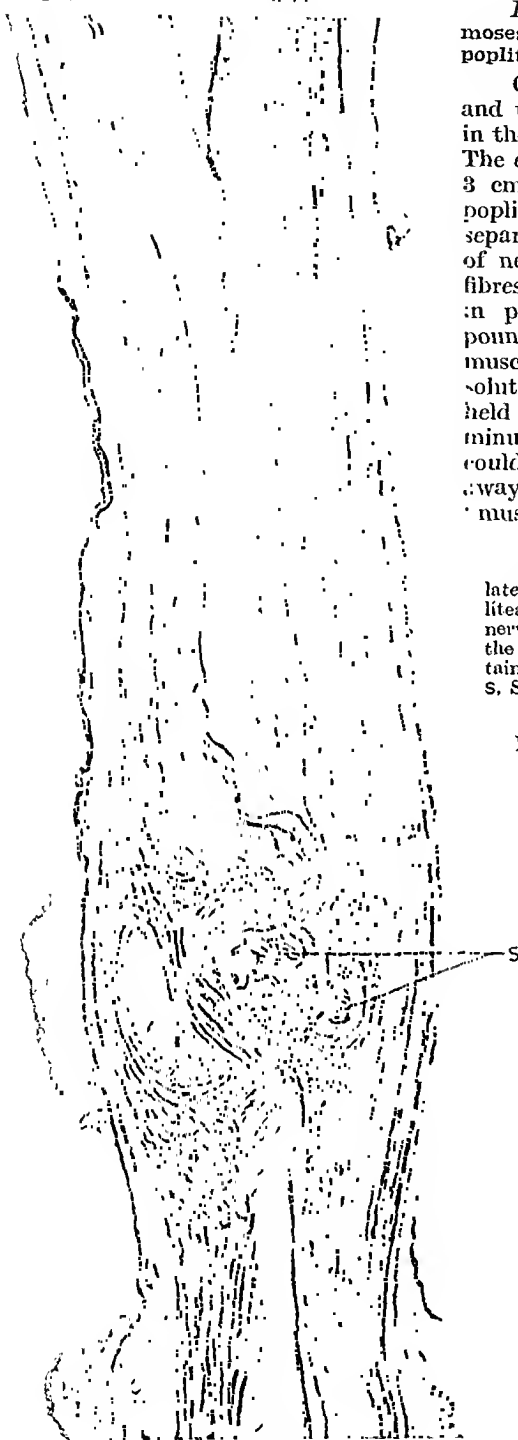


FIG. 362.

*Experiment 4.*—Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.

*Cat.*—External popliteal nerve divided and the ends implanted into incisions made in the middle of the internal popliteal nerve. The ends of the external popliteal nerve were 3 cm. apart. The incisions in the internal popliteal nerve appeared to be more like separations of nerve bundles than divisions of nerve-fibres: it was doubtful if nerve-fibres were cut. The anastomoses were kept in place by 'muscle varnish', made by pounding in a mortar a small piece of living muscle with a few drops of sterilized salt solution. This produced a sticky mass which held the nerves together. After a few minutes the 'varnish' was adherent and could be pulled on by forceps without coming away. This was the first occasion on which 'muscle varnish' was used.

*FIG. 362.*—*Experiment 3.* Monkey. Double lateral implantation of the divided external popliteal nerve into the side of the internal popliteal nerve. The figure shows the upper anastomosis; the lower anastomosis was lost. Specimen obtained 10½ months afterwards. Cajal stain. S, Site of suture. ( $\times 10$ .)

1½ months.—Wound opened; external popliteal nerve-ends in place; a good deal of new tissue, red and vascular, around each anastomosis.

*Results of Faradic Stimulation*  
(*Fig. 363*):—

- A and B, No extension.
- C, Sharp flexion.
- D, A little less flexion.
- E, Very slight flexion.

The same strength current used in each case.

3 months.—No lameness in walking. This is an aerobatic cat; climbs anywhere; there is a wire netting near the ceiling of the room in which the cats were kept. This animal would spring from a ledge to this netting and walk along upside down, there being no appearance of weakness of the operated limb. On holding the cat up by the shoulders there was not so much dorsiflexion of the ankle of the operated limb as of the ankle of the normal limb: but the claws of both limbs were equally exposed. (Sir David Ferrier was present.)

5 months.—Claws and ankle extended strongly.

10 months.—Animal etherized and subsequently killed with ether.

### Faradic Stimulation through Skin :—

1. At the back of thigh : Flexion of ankle and toes and protrusion of claws (? extension) occurred.
2. Over upper and outer part of leg, over region of external popliteal nerve : Extension of ankle, toes, and claws.

The anastomoses were exposed and the nerves stimulated with the faradic current.

### Results of Stimulation (Fig. 364) :—

- A and B, Extension of ankle, toes, and protrusion of claws.
- E, Extension of ankle, toes, and protrusion of claws.
- C, Flexion of knee, ankle, toes, and protrusion of claws.
- D, Flexion of ankle, toes, and protrusion of claws.
- F, Variable : sometimes flexion, sometimes extension, as if internal popliteal nerve-fibres were joined to both nerves distal to this point of stimulation.

At B and D more current was required to produce the same effect than was necessary at A and C.

**Microscopical Appearances.**—Fig. 365 shows the position of the two ends of the external popliteal nerve within the internal popliteal nerve. Fig. 365, C shows the regeneration of fibres in the distal segment of the external popliteal nerve.

FIG. 363.—Experiment 4. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 1½ months.

Fig. 366, A is an enlarged view of the bulb at the end of the proximal segment, and shows the multitude of new fibres present. Fig. 366, B is an enlarged picture of the proximal end of the distal segment. Without examining serial sections it is not easy to follow the anatomical connections of the two ends of the external popliteal nerve with the columns of fibres on either side and beyond them.

**Experiment 5.**—Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.

**Cat.**—External popliteal nerve divided and the ends united to transverse incisions made in the side of the internal popliteal nerve, 4½ cm. apart.

1½ months.—Animal walking well. The toes were slightly flexed when at rest as compared with those on the normal side. Both extension and flexion of the ankle were strong movements.

At this date the animal was killed by ether inhalation, but before death occurred the anastomoses were exposed and stimulated with the faradic current. (Sir David Ferrier was present.)

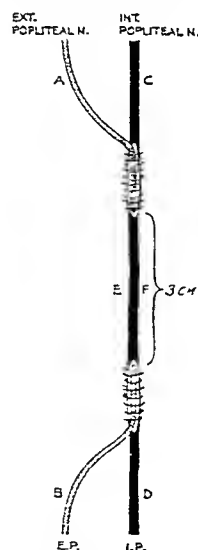


FIG. 364.—Experiment 4. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 10 months.

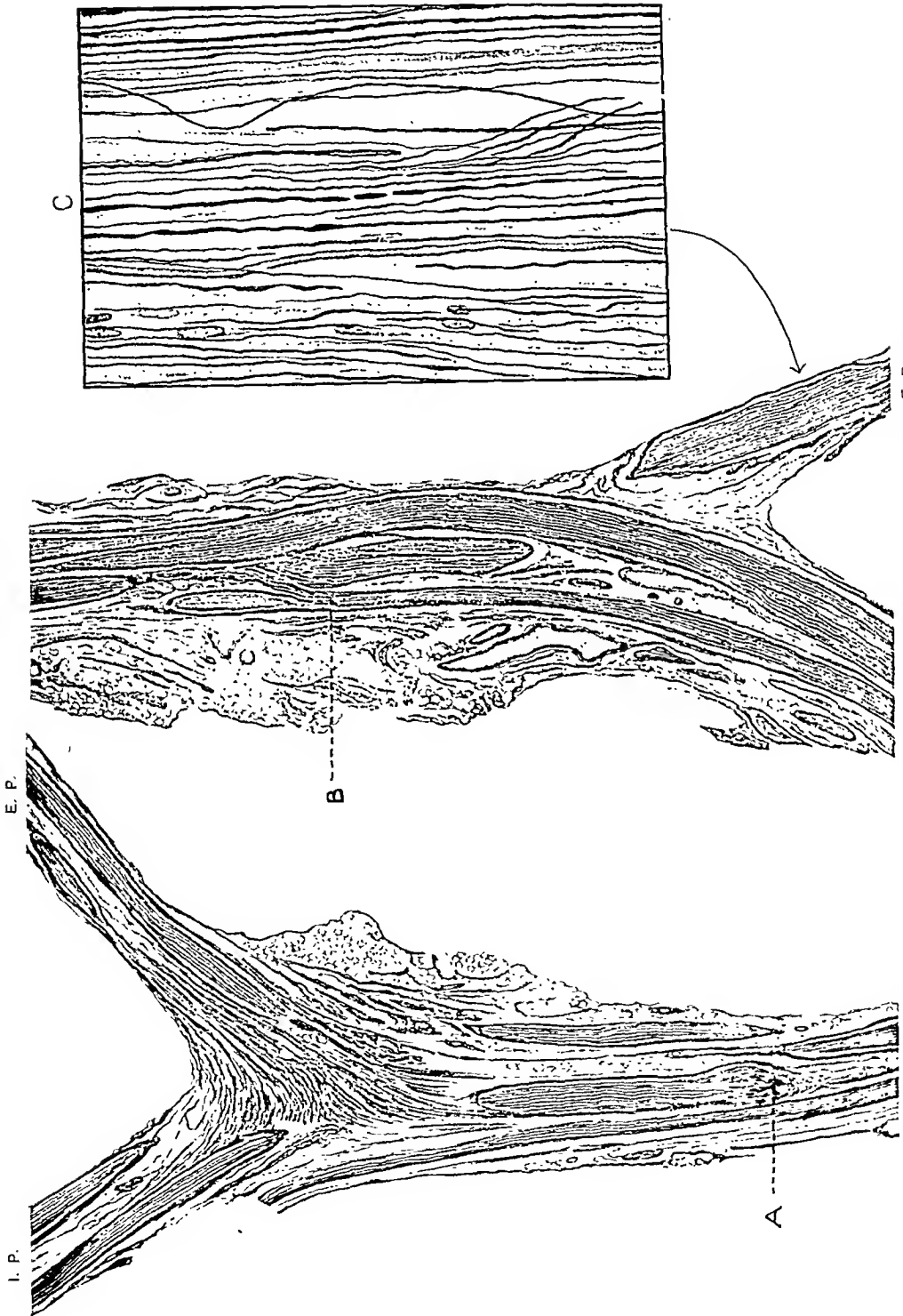
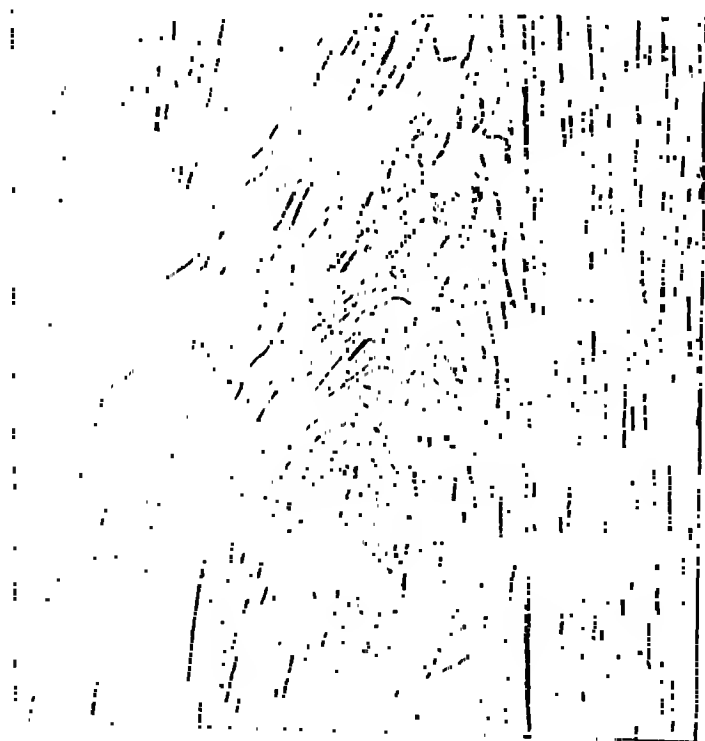


FIG. 205.—*Experimental A.* Cat. External popliteal nerve divided, and the ends implanted into the middle of the nerve. *B.* Same cat. 10 months afterwards. ( $\times 75$ ). *C.* is a more highly magnified ( $\times 450$ ) portion of the nerve. *A* and *B* indicate portions more highly magnified in *Figs. 206* and *207*.



A



B

FIG. 366.—A, Bulb (somewhat compressed) of distal end of proximal segment of external popliteal nerve shown at A in *Fig. 365*. ( $\times 100$ .) B, Proximal end of distal segment of external popliteal nerve shown at B in *Fig. 365*. ( $\times 45$ .)

*Results of Stimulation (Fig. 367):—*

- A, Extension of ankle (strong) and marked protrusion of claws.
- B, Flexion of ankle; with full flexion claws were exposed.
- C, Flexion of toes. Flexion of ankle, but not such a strong movement as at B.
- D, Flexion of ankle. Strong flexion of toes; claws not protruded.
- E, Strong flexion of ankle and slight flexion of toes.

The transverse incisions in the internal popliteal nerve extended across the nerve for half its diameter. It would seem that the upper segment of the external popliteal nerve was at this stage of the experiment (4½ months) chiefly connected with fibres of the internal popliteal nerve. The flexion movement is the stronger, and it might have masked a weak extension movement when E was stimulated: or perhaps it was too soon for functional union to have taken place between the proximal and distal segments of the external popliteal nerve. The striking fact was that when A (distal segment of external popliteal nerve) was stimulated, strong extension of ankle and toes occurred, so this part of the external popliteal nerve had reached the stage of regeneration in which it exhibited the phenomenon of excitability.

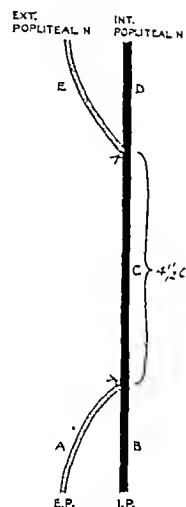


FIG. 367.—*Experiment 5.* Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 4½ months.

*Experiment 6.*—Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.

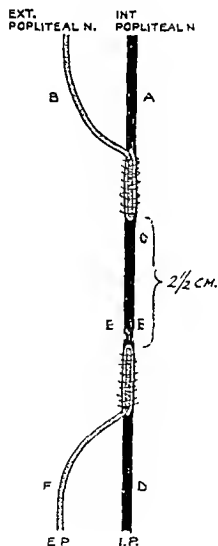


FIG. 368.—*Experiment 6.* Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 9 months.

CAT.—External popliteal nerve divided and the ends inveigled into longitudinal median incisions in the internal popliteal nerve, 2½ cm. apart. The anastomoses were fixed with 'muscle varnish'.

3 months.—Walks well. On being held up by the shoulders the ankle is extended and the claws protruded, but not so much as on normal side.

7 months.—Ankle extended and claws protruded as on normal side.

9 months.—Animal was ill, and was killed by ether inhalation. Before death the nerves were exposed and stimulated with the faradic current.

*Results of Stimulation (Fig. 368):—*

- A, Strong flexion of toes.
- B, Combined movement. Contraction both of extensor and flexor muscles.
- C, Less strong flexion of toes.
- D, Only slight flexion of toes.
- E, E, Extension of ankle and toes.
- F, " " " " " " but not so strongly marked as at E, E.

*Experiment 7.*—Double lateral anastomoses of the divided ends of the external popliteal nerve to internal popliteal nerve.

CAT.—External popliteal nerve divided and the ends inveigled into longitudinal median incisions made in the internal popliteal nerve, 3 cm. apart. The anastomoses were fixed by 'muscle varnish'.



3 months.—Only slight extension of ankle and toes on being held up by the shoulders.

At this date the animal was ill, and was killed by ether anæsthesia. Before death the nerves were exposed and stimulated with the faradic current. (Stimulation of the extensor muscles through the skin produced no response.)

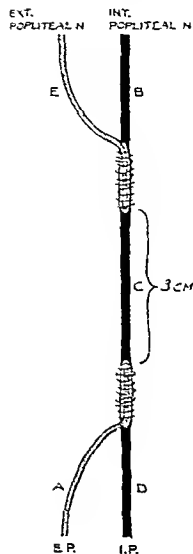


FIG. 369.—Experiment 7. Diagram showing the points on the popliteal nerves at which faradic stimulation was applied. 3 months.

movements were the flexion of the metacarpophalangeal joints by the lumbricales muscles and the drawing inwards of the thumb over the palm by the adductor and the opponens pollicis muscles.

4 months.—*Faradic stimulation.* Response is present in all the intrinsic muscles supplied by the median nerve. The intrinsic muscles supplied by the ulnar nerve—the interossei, the two inner lumbricales, the muscles of the hypothenar eminence, and the deep intrinsic muscles of the thumb—all contract, but the response to stimulation is not so easily obtained as is the response of the intrinsic muscles supplied by the median nerve.

5 months.—*Faradic stimulation.* Response of all the intrinsic muscles of the hand obtained with more ease. The hand is being used for all purposes, but when food is offered the undamaged hand takes it.

8½ months.—Response to galvanic current in all intrinsic of hand normal. Response to weak normal faradic current is easily obtained in all the muscles.

10 months and 10 days.—The monkey was killed by ether inhalation. Before death the anastomoses were exposed and the nerves stimulated with the faradic current.

#### Results of Direct Stimulation (Fig. 369):—

- A, Extension of ankle and toes.
- B, Strong flexion of ankle and toes.
- C, Less strong flexion of ankle and toes.
- D, Only slight flexion of ankle and toes.
- E, Combined flexion and extension movements: the flexion movement being the most powerful.

*Experiment 8.*—Double lateral anastomoses of the divided ends of the ulnar nerve to the median nerve.

*RHESUS MONKEY.*—Double lateral implantation of divided ulnar nerve into the side of the median nerve, 1½ in. apart. The median nerve was incised for ½ in. (or more) at the site of each anastomosis. The ends of the divided ulnar nerve were passed under the superficial muscles and tendons of the forearm to reach the anatomical position of the median nerve. One suture of Van Horn arterial iron-dyed silk was used to fix each anastomosis. The attempt was made to pass each suture through the sheath of the nerves only. The median and ulnar nerves (distal portions) were stimulated with the faradic current before the anastomoses were carried out. These stimulations caused contraction of the intrinsic muscles of the hand supplied by each nerve. The most striking

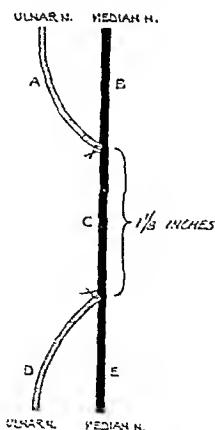


FIG. 370.—Experiment 8. Diagram showing the points on the ulnar and median nerves at which faradic stimulation was applied. 10 months and 10 days.

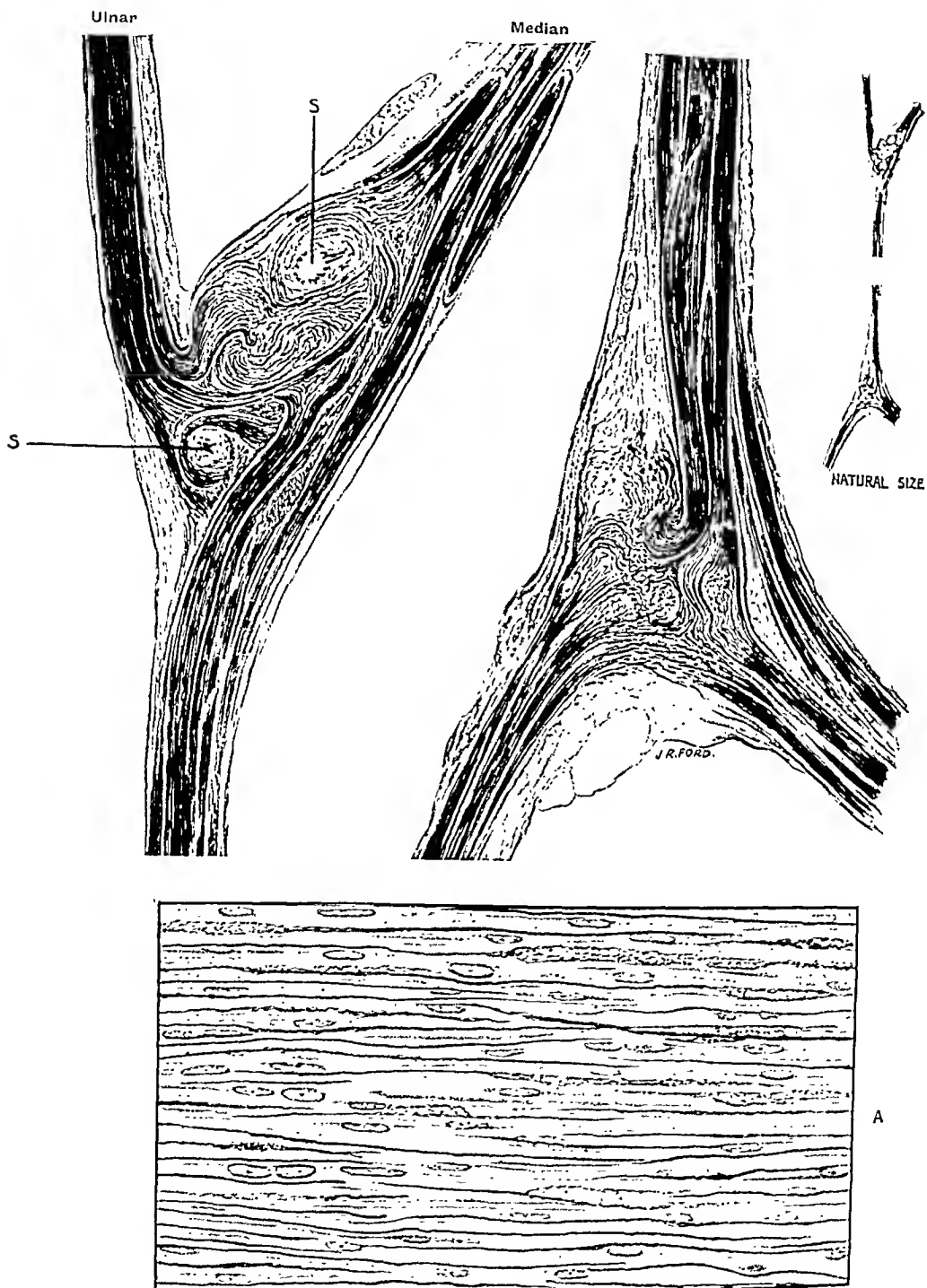


FIG. 371.—Experiment 8. Monkey. Double lateral implantation of divided ulnar nerve into the side of the median nerve,  $1\frac{1}{2}$  in. apart. Specimen obtained 10 months and 10 days afterwards. Weigert stain. S, Site of suture. ( $\times 7$ .) A, High-power view of distal part of ulnar nerve. Stroebe stain. ( $\times 450$ .)

*Results of Stimulation (Fig. 370):—*

- A, Response of median intrinsics.  
 B, " " ulnar  
 C, " " both median and ulnar intrinsics.  
 D, " " median intrinsics, weaker movement than at A (same current).  
 E, " " ulnar intrinsics, weaker movement than at B (same current).

*Microscopical Appearances.*—The microscopical specimens were stained by the Weigert and Stroebe methods. In Fig. 371 the appearances show that the median nerve was considerably injured by the transverse incision made at the operation. There is no new tissue on the ulnar side of the main trunk of the median nerve, as is seen in a corresponding position when this type of operation is performed on the popliteal nerves; but in some of the sections this tissue is visible and contains columns of new nerve-fibres. S shows the site of the remains of one of the sutures. Fig. 371, A is a high-power view of the regenerated distal segment of the ulnar nerve (the Stroebe stain showing the new axis cylinders). The new fibres joining the ends of the ulnar nerve to the median nerve are very numerous, and group themselves in various curving bands of fibres which appear definitely to join with corresponding groups of median nerve-fibres. The right forearm of the animal was operated on, but, in the process of mounting, the sections were turned over so that in the figure the nerves are placed as in the left forearm. The lateral spreading out of the bands of fibres of which the median nerve is composed is due to the tension on the tissues of the nerve when pinned out on a sheet of cork during the hardening process.

*Experiment 9.*—Double lateral anastomoses of the divided ends of the median nerve to the ulnar nerve.

*Rhesus Monkey.*—Double lateral implantation of divided median nerve of forearm into the side of the ulnar nerve,  $1\frac{1}{2}$  in. apart. The ulnar nerve at the site of the proximal anastomosis was divided almost completely, while at the site of the distal anastomosis about one-half of the nerve was divided. The ends of the median nerve were passed under the superficial muscles and tendons of the forearm to reach the anatomical position of the ulnar nerve. One suture of Van Horn arterial iron-dyed silk was used to fix each anastomosis. The natural tension of the living nerve caused the incisions in the ulnar nerve to gape considerably.

Before the experiment was commenced both nerves were stimulated with the faradic current. *Median nerve stimulation:* Flexion of the metacarpal phalangeal joints of the 1st and 2nd fingers, and the thumb was drawn across the palm (opponens pollicis). *Ulnar nerve stimulation:* Flexion of 3rd and 4th fingers at the metacarpal phalangeal joints, and adduction of thumb. The other intrinsic muscles of the hand contracted, but the above movements were the most important visible effects of stimulation of these two nerves.

$4\frac{1}{2}$  months.—*Faradic stimulation of muscles.* Response observed in all the intrinsic muscles of the hand, including the two outer lumbricals.

$5\frac{1}{2}$  months.—Same report.

9 months.—*Faradic stimulation.* All the median intrinsics respond to a weak current.

$10\frac{1}{2}$  months.—Monkey was ill, and was killed by ether inhalation. Before death the anastomoses were exposed and the nerves stimulated with the faradic current.

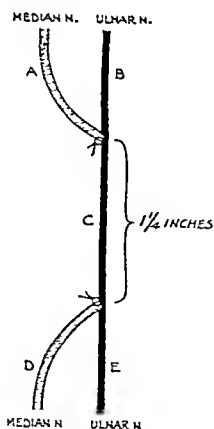


FIG. 372.—*Experiment 9.* Diagram showing the points on the median and ulnar nerves at which faradic stimulation was applied.  $10\frac{1}{2}$  months.

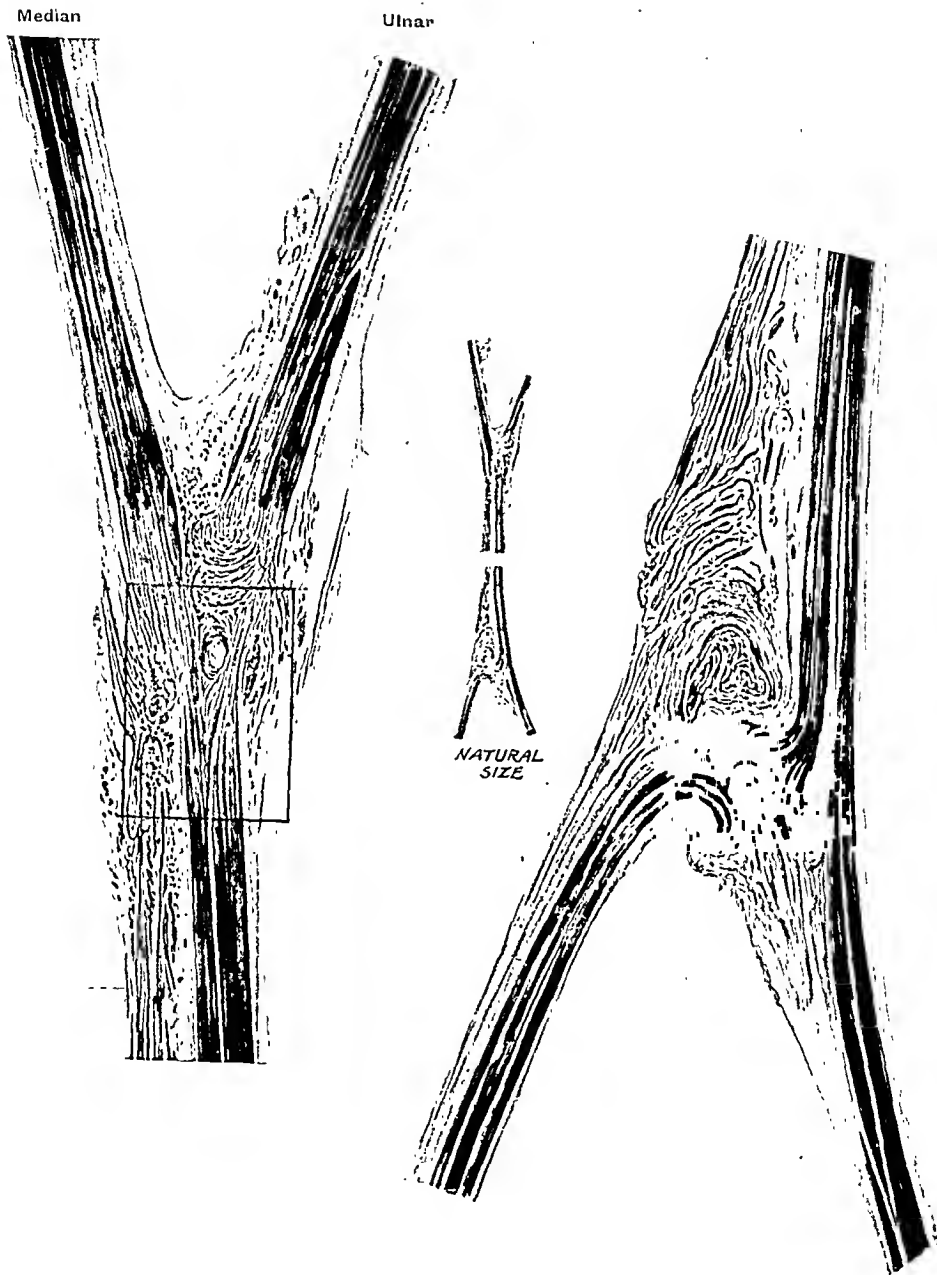


FIG. 373.—*Experiment 9. Monkey. Double lateral implantation of divided median nerve of forearm into the side of the ulnar nerve,  $1\frac{1}{2}$  in. apart. Specimen obtained 10½ months afterwards. Weigert stain. The black rectangle shows the part included in Fig. 371, A. ( $\times 7$ .)*



FIG. 374.—A, More highly magnified view of part included in black rectangle in Fig. 373. Upper anastomosis, showing fibres of ulnar nerve continuous with median nerve at site of suture. 1, New median nerve-fibres external to main ulnar trunk; 2, Part of ulnar nerve continuous at site of suture with both median and ulnar fibres; 3, The other half of ulnar nerve not communicating with median nerve. ( $\times 30$ .) B, High-power view of the part included in the black rectangle in Fig. 373, 2. Stroebe stain. ( $\times 300$ .)

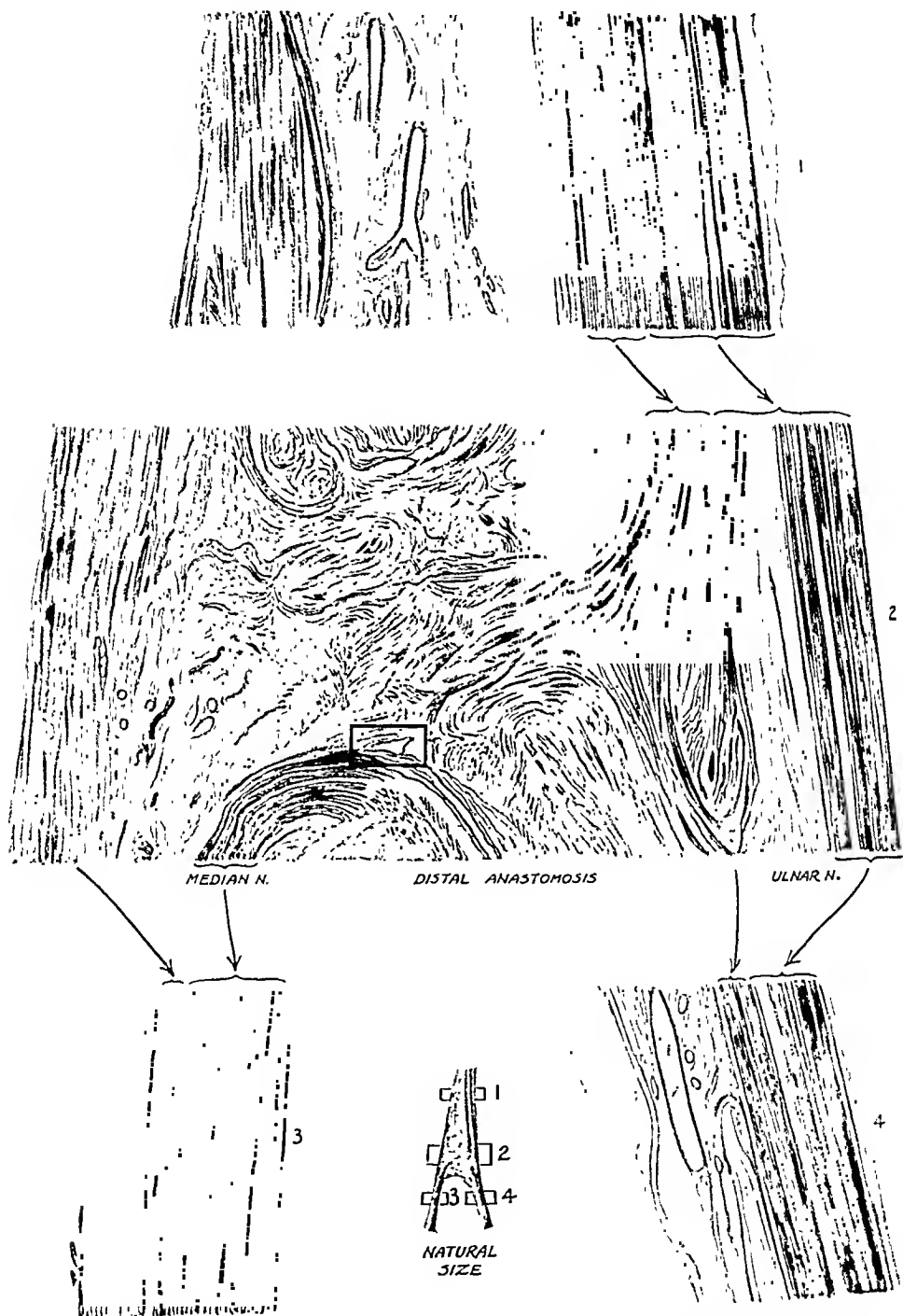


FIG. 375.—Higher-power view of the parts in the neighbourhood of the distal anastomosis shown in Fig. 373. 1 shows, on the right, part of the ulnar nerve above the anastomosis isolated by incisions into which the divided ends of the median nerve were sutured; this part has regenerated, and through it impulses pass from the proximal to the distal portions of the median nerve; on the left is seen the tissue external to the nerve between the two anastomoses. 2 shows the distal anastomosis itself; the part included in the black rectangle is shown under higher magnification in Fig. 374, B. 3 and 4 show the median and ulnar nerves respectively below the anastomosis. Stroebe stain. ( $\times 30$ .)

*Results of Stimulation (Fig. 372):—*

A. Response of median intrinsics.	1	Weak current.
B. " " ulnar "	1	
D. " " median intrinsics	1	Current stronger than required
E. " " ulnar "	1	at A and B.
C. No response.		

*Microscopical Appearances.*—*Fig. 373* gives a general view of the anastomosis.

*Fig. 374, A* is an enlarged view of the proximal anastomosis. The part selected for the drawing is marked by lines in *Fig. 373*. The course of the fibres at the anastomosis is indicated in the legend below the drawing. The wide band of new tissue on the median side of the ulnar nerve, occupying the region between the two transverse incisions which were made in the side of the ulnar nerve at the operation, is seen to be traversed by new sheaths which are connected with the proximal end of the median nerve.

*Fig. 375* includes sections stained by the Stroebe method to show the distal anastomosis, the median and ulnar nerves above and below the distal anastomosis, and the nerve-fibre anastomoses. It would appear that the nerve-fibres of the distal segment of the median nerve have formed three connections: (a) With nerve-fibres in the new tissue on the median side of the main ulnar nerve-trunk; (b) With a band of nerve-fibres of the main ulnar nerve-trunk; and (c) With fibres of the ulnar nerve beyond the distal anastomosis.

*Fig. 374, B* is a high-power drawing of a portion of the drawing marked 2 in *Fig. 375*, stained by the Stroebe method. The part selected for the drawing is marked by lines. The rod-shaped nuclei of numerous neurilemma cells cover the whole field. They lie in the same direction as the developing young axis cylinders. The neurilemma cells are spindle shaped, and form long lines of cells with the processes in contact or overlapping.

The young axis cylinders, when examined with a Zeiss  $\frac{1}{12}$  oil immersion lens and ocular 2, appear to lie in the protoplasm of the neurilemma cells. When a Weigert-stained specimen is examined under the same magnification, the young sheaths lie also in the protoplasm of the neurilemma cells. The impression produced is that both axis cylinders and sheaths are developed from the protoplasm of the neurilemma cells.

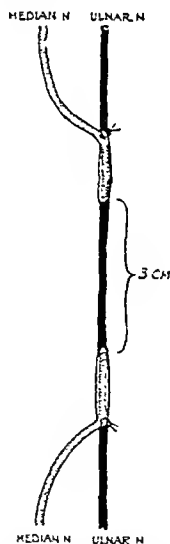
*Experiment 10.*—Double lateral anastomoses of the divided ends of the median nerve to the ulnar nerve.

**RHESUS MONKEY.**—Median nerve of forearm divided, and the ends, after being passed under the flexor muscles and tendons, inveigled into median incisions made in the trunk of the ulnar nerve, 3 cm. apart. Half an inch from each end of the median nerve a fine silk suture was passed through the sheaths of both median and ulnar nerves (*Fig. 376*). In this manner the fixing sutures were each some distance from the cut ends of the median nerve. No 'muscle varnish' was used.

*4 months.*—*Faradic stimulation.* Moderate strong current: All the median intrinsics contracted, but the 2nd lumbrical showed only a weak movement. Moderate current: Same report.

*8 months.*—Median and ulnar intrinsics all react to a weak current. A film of the movements was taken.

The experiment is not yet complete.

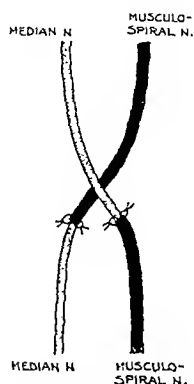


**FIG. 376.**—*Experiment 10.* Diagram showing ends of divided median nerve inveigled into median incisions in trunk of ulnar nerve.

All the ulnar intrinsics contracted, including all the interossei and the 2 inner lumbricals.

*Experiment 11.—Crosswise anastomoses of the median and musculospiral nerves.*

**BABOON.**—The median and musculospiral nerves were divided in the upper arm region. The proximal part of the musculospiral was sutured to the distal segment of the median nerve and the end of the proximal segment of the median nerve was sutured to the distal part of the musculospiral nerve. Two sutures of Van Horn arterial silk were used to fix each anastomosis (*Fig. 377*). A piece of fascia lata was wrapped round each anastomosis so as to prevent the anastomoses coming into contact.



**FIG. 377.**—*Experiment 11.* Diagram showing crosswise anastomoses operation of the median and musculospiral nerves.

**1½ months.**—*Faradic stimulation.* No movement observed in muscles supplied by the median and musculospiral nerves distal to the anastomoses.

**5 months.**—*Faradic stimulation.* Front of forearm: Good response observed in flexor carpi radialis and in flexor sublimis and flexor profundus digitorum. No response in intrinsic muscles of hand supplied by median nerve. Extension: Good response in triceps and supinator longus only.

**10½ months.**—Unfortunately the baboon died from acute pneumonia before any further examination of the muscular recovery had been made. The microscopical examination of the anastomoses in the upper arm and of the posterior interosseous and median nerves in the upper

part of the forearm show that considerable nerve regeneration had taken place.

*Microscopical Appearances.*—*Fig. 378, A* shows the median-musculospiral anastomosis 10½ months after operation. The distal end of the proximal segment of the median nerve is bulbous. The regenerated fibres in the musculospiral nerve below the anastomosis are finer and take the stain less deeply than the fibres of the median above the anastomosis.

*Fig. 378, B* shows the musculospiral-median anastomosis. The distal end of the proximal segment of the musculospiral nerve is bulbous. The fibres of the regenerated median nerve below the anastomosis take the stain less deeply below the anastomosis than the fibres of the musculospiral nerve above the anastomosis.

Nerve-fibres pass through both anastomoses, thus explaining the functional results observed.

*Fig. 379, A* shows regeneration of neurilemma sheaths in the median nerve below the bend of the elbow, while *Fig. 379, B* shows regenerated axis cylinders in the posterior interosseous nerve below the bend of the elbow.

*Experiment 12.—Crosswise anastomoses of the internal and external popliteal nerves.*

**RHESUS MONKEY.**—The external and internal popliteal nerves were divided. The proximal segment of the internal popliteal nerve was united to the distal segment of the external popliteal nerve, and the proximal segment of the external popliteal nerve was united to the distal segment of the internal popliteal nerve (*Figs. 380, 381*). A thick piece of muscle was placed between the anastomoses. Two sutures of Van Horn arterial silk was used to fix each anastomosis.

**2 months.**—Wasting of leg muscles. No reaction to weak faradic current.

Response to strong faradic current: Tibialis anticus, extensor communis digitorum, and peronei; all flexor muscles of leg, and the abductor pollicis of the foot. Response to galvanic current: Slow contraction of all muscles.

**4 months.**—Animal ill, hence was killed by ether anaesthesia. Before death the nerves were exposed and stimulated with the faradic current.



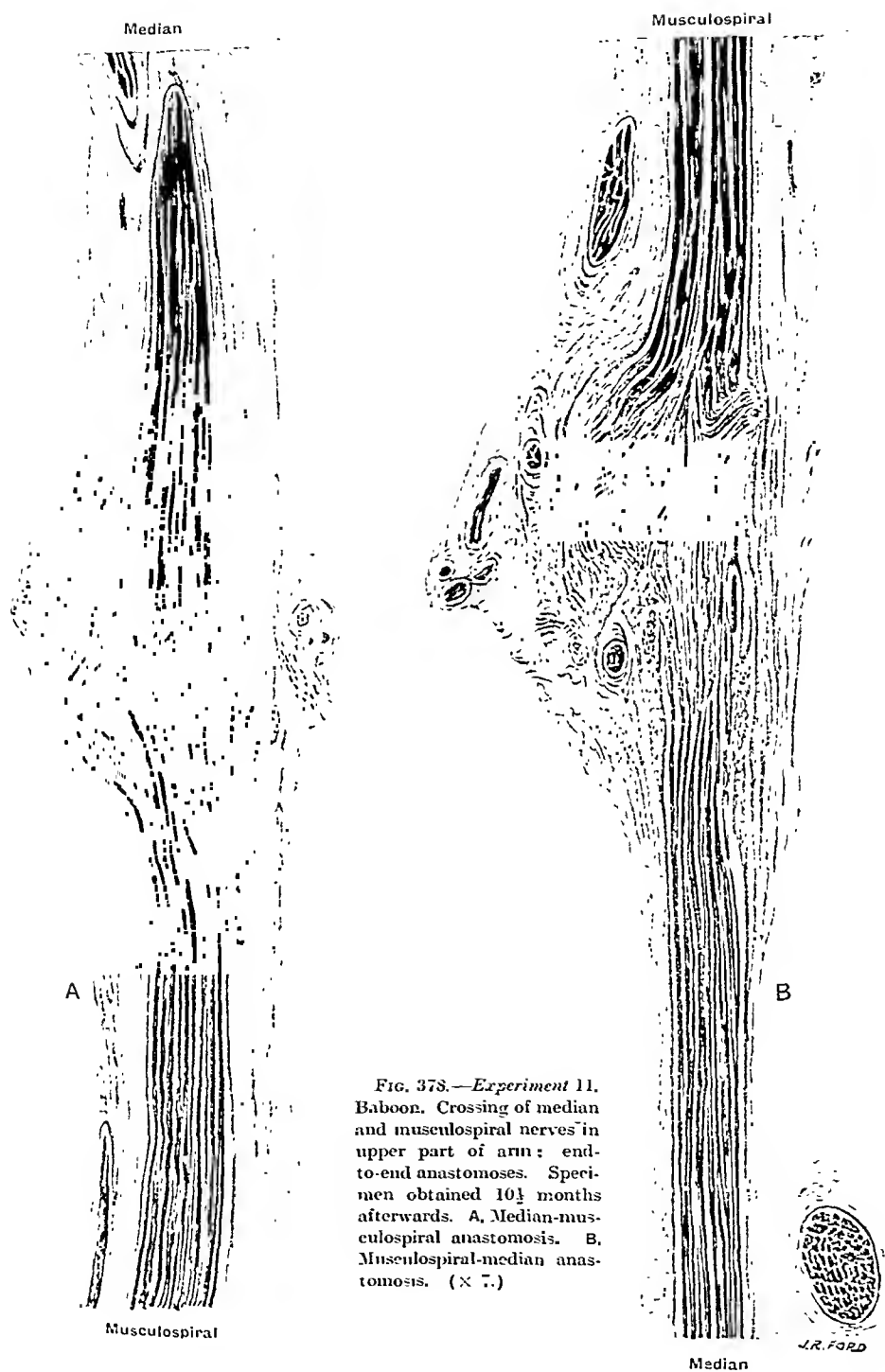
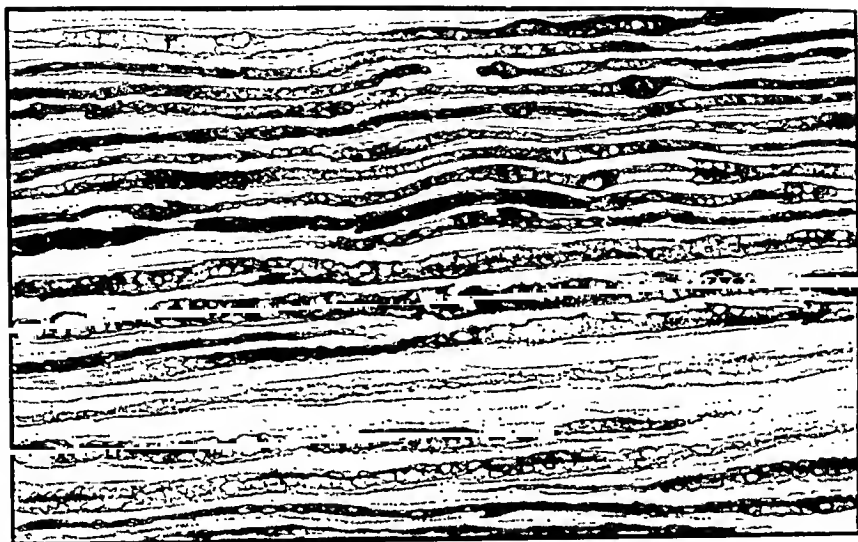
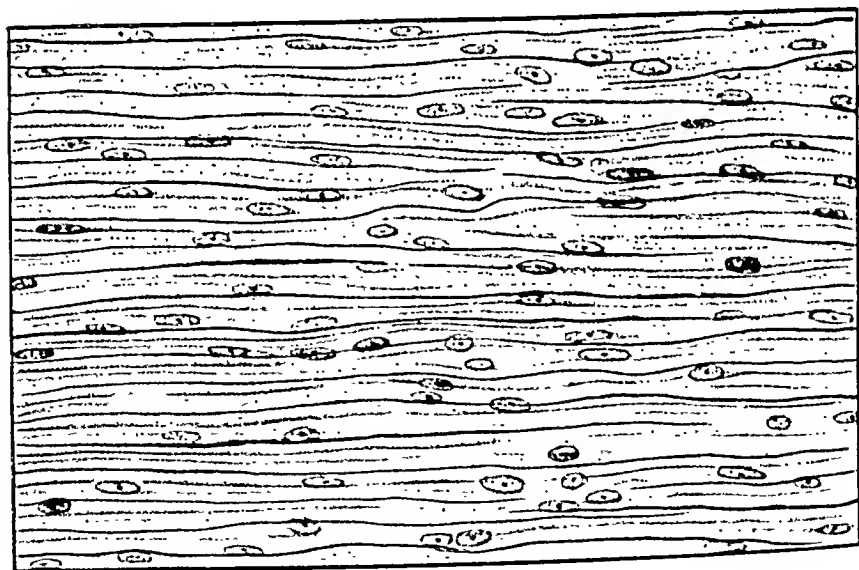


FIG. 378.—*Experiment 11.* Baboon. Crossing of median and musculospiral nerves in upper part of arm: end-to-end anastomoses. Specimen obtained 10½ months afterwards. A, Median-musculospiral anastomosis. B, Musculospiral-median anastomosis. ( $\times 7$ .)



A



B

FIG. 379.—From the same experiment as *Figs. 377 and 378*. Portions of the median nerve (A) and the posterior interosseous nerve (B) removed below the bend of the elbow. The sections show regeneration of both nerves. A was stained with Weigert, B with Strocker. ( $\times 450$ .)



FIG. 380.—Experiment 12. Monkey. Crossing of internal and external popliteal nerves in the popliteal space: end-to-end anastomoses. Specimen obtained 4 months afterwards. ( $\times 7$ .)

*Results of Stimulation (Fig. 382):—*

A. Flexion of toes and ankle.

B. Strong extension of ankle. The toes were curled up owing to shortening of the flexor tendons and muscles, but the extensors of the toes contracted.

The monkey died before stimulation of the appropriate areas of the opposite Rolandic cortex for extension and flexion of the ankle could be carried out.

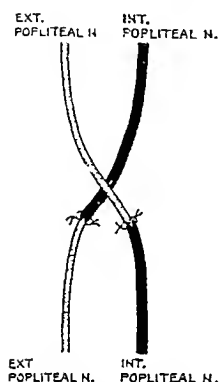


FIG. 381.—*Experiment 12.* Diagram showing crosswise anastomoses operation of the internal and external popliteal nerves.

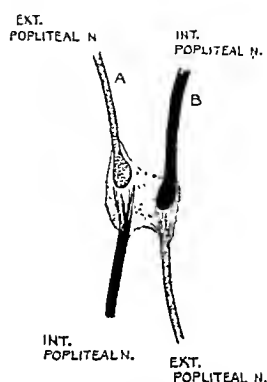


FIG. 382.—*Experiment 12.* Diagram showing the points on the popliteal nerves at which faradic stimulation was applied, and also the parts removed by dissection after death, 4 months.

*Microscopical Appearances.*—*Fig. 380* reveals the fact that union has taken place between the external popliteal and internal popliteal nerves and between the internal popliteal and external popliteal nerves respectively at the sites of the two anastomoses. The distal ends of the proximal segments of the external and internal popliteal nerves are both bulbous. In the regenerated segments of both popliteal nerves distal to the anastomoses the fibres are finer, more numerous, and less deeply stained than the fibres in the nerves proximal to the anastomoses.

## ILEUS FOLLOWING FRACTURED RIBS.

BY F. G. RALPHS, DUKINFIELD.

UNCOMPLICATED fracture of the ribs is a surgical commonplace; its association with grave abdominal symptoms is of sufficient rarity to make it worthy of record.

The patient was a store-keeper, age 60, who was admitted to hospital with the following history. Six days previously he had slipped on a frosty pavement, falling heavily on his right side. He was conveyed home, and strapping was applied to the chest by his doctor. In the ensuing days, the bowels failed to respond to the usual aperients, though prior to the injury he had never been subject to constipation and was accustomed to a daily stool. The day before admission it was noticed that his abdomen was distended, and he was removed to hospital.

On removing the strapping, crepitus and surgical emphysema were noted in the right axilla. A skiagram revealed ununited fractures of the sixth and seventh ribs midway between their angles and sternal ends. There was general distention of the abdomen, with a tympanitic note on percussion. The distention was most marked centrally. Neither tenderness nor rigidity was present, and rectal examination proved negative. The tongue was thickly furred and dry, temperature normal, pulse 90. An enema gave a small faecal result with the passage of flatus, but the distention persisted despite the administration of pituitrin and eserine. By the following day, his condition had obviously deteriorated. The distention had increased, the pulse was quick and weak, and another enema failed to relieve the absolute constipation, now of twenty-four hours' duration.

The condition had apparently developed into one of acute obstruction, and operation was decided upon. His transit to the theatre was interrupted by a copious vomit of dark-brown intestinal contents, of faecal odour. Under novocain anaesthesia, enterostomy was quickly performed through a median incision, two inches in length, just above the umbilicus. The base of the first distended loop of small intestine which presented was sutured to the parietal peritoneum, and the gut opened. There was an instant escape of flatus and gush of liquid faeces, soiling the wound thoroughly before a catheter could be tied in the bowel for drainage.

During the next twelve hours, eighteen ounces of fluid faeces drained away, and as the abdominal distention persisted, another enema was given, with the expulsion of much flatus. On the second day after operation, the patient had four normal motions per rectum, there being no drainage through the enterostomy tube. On the third day, eighteen ounces escaped through the tube, the bowels did not move naturally, but much flatus was passed after a turpentine enema. By the evening of the third day the distention

had completely disappeared. On the fourth day the patient plucked the tube from his wound without untoward result, the fecal fistula draining freely into the dressings. About this time he exhibited restlessness and mental confusion. From the fifth day onwards the bowels were moved naturally, and the amount of drainage from the fistula steadily diminished. Five weeks after operation it was completely closed, and the wound soundly healed.

During convalescence a systematic examination of the gastro-intestinal tract was carried out by opaque meal and enema. No lesion was found to account for the symptoms of obstruction, nor was any delay noted in the passage of the meal through the intestines. Hence the condition which supervened has been regarded as one of paralytic ileus. Since discharge from hospital sixteen months ago the man has remained steadily at work and enjoyed good health without recourse to aperients.

OBSERVATIONS.—A search of the literature has revealed a single reference to a like condition following upon rib fracture. Some years ago, Mr. J. E. Adams,<sup>1</sup> in a paper, "Paralytic Ileus as a Sequel of Fractured Ribs", described two cases similar to the above, in one of which he employed the same method of treatment by ileostomy. In both the fracture was close to the angles of the ribs, and was therefore in the region of the sympathetic cord and ganglia lying on the necks of the ribs. Arguing from this relation, Adams inferred that "some displacement of the proximal fragments occurred sufficient to irritate the sympathetic chain", and, *pari passu*, the splanchnic nerves, arising from the lower half of the gangliated cord in the thorax. Now Starling<sup>2</sup> has shown that stimulation of these nerves produces inhibition of peristalsis in the small intestine. To splanchnic irritation, then, was ascribed the train of abdominal symptoms which occurred in the two previously reported cases. It is an ingenious theory, but difficult of comprehension from the anatomical standpoint. The head of the rib is so securely anchored by short and strong ligaments as to prohibit, one must think, the range of movements assumed for it by the hypothesis above. As stress has been laid by Adams on the site of the fracture, it is of interest to note that in the present case it was situated at some distance from the angle, viz., in the axilla.

Again, cases of multiple fracture of ribs are not uncommonly found in the accident wards of hospitals. Yet how infrequent is the occurrence of the abdominal condition outlined in this case! Perhaps some other explanation might be attempted. Maybe the rib fracture and the intestinal paralysis are alike the results of a common cause, i.e., the accident, and not dependent the one upon the other. Now it is known that, with the splanchnics intact, inhibition of the movements of the small intestine follows on stimulation of the sensory nerves of the abdomen. An every-day demonstration occurs to us in the temporary inhibition of peristalsis which follows an abdominal section. A slight distention of the intestine with gas results, and usually begins to pass off in twelve to twenty-four hours after operation. Should it persist, the conditions would be favourable for the onset of paralytic ileus. It may be argued in the cases under consideration that a similar inhibition of peristalsis is produced through irritation of the abdominal sensory nerves by the injury.

To explain why this paralytic condition should endure, and terminate in ileus, some other factor seems necessary. Perhaps it is a vascular one, not peculiar to the territory of the intestines, but involving the systemic circulation as a whole. A review of the clinical features of these three cases shows that in two of them a note was made of the altered mental condition of the patient. Thus, in the first case, reported by Adams, it is described as "abnormal", and "mental depression was still a marked feature of the man's condition". In the present case, similar mental changes have already been described. This simultaneous exhibition of cerebral and abdominal symptoms leads one to suggest that, following upon the rib fracture, traumatic lipæmia and fat embolism may have been a causal factor in the production of the sequelæ described. Of the presence of this in the case under consideration no evidence can be adduced, from the regrettable omission to make systematic examination of the blood and urine during the man's stay in hospital. Thus it is merely speculative, and presented, with all reserve, as an explanation of the perplexing symptoms which may rarely follow simple fracture of the ribs.

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REFERENCES.

- <sup>1</sup> ADAMS, J. E., "Paralytic Ileus as a Sequel of Fractured Ribs," *Ann. of Surg.*, 1910, II, 102.
- <sup>2</sup> STARLING, *Recent Advances in the Physiology of Digestion*, 1905.

## PLASTIC OPERATION FOR COVERING A GUILLOTINE AMPUTATION STUMP.

By W. B. GABRIEL, LONDON.

THE methods at present recognized for treating a guillotine amputation stump prior to healing are either: (1) Glue or strapping extension on soft parts, which usually results in a puckered and adherent terminal scar; or (2) Re-amputation if the bone has failed to be covered by the previous method.

The covering of a guillotine stump by a pedicled graft is here suggested as a better method, in that it avoids the necessity of re-amputation and gives a much firmer stump than can possibly be obtained otherwise without sacrificing a further length of bone.

It is an established principle (Huggins<sup>1</sup>) that in a forearm stump at least three inches and sometimes four inches of ulna are necessary to enable a forearm bucket to be fitted. In the case now to be described with the aid of the accompanying photographs, the employment of a pedicled graft enabled a useful length of a forearm stump to be saved, and provided a firm covering of healthy skin and fascia over the end of the stump. The case was one in which it was particularly important that as much bone as possible should be conserved, seeing that both hands had been lost. Reference to *Fig. 383* shows that it would have been impossible in the ordinary way to cut adequate flaps and still preserve the necessary length of bone below the elbow to enable an appliance to be fitted.

The patient, a male, age 21, was admitted to the Middlesex Hospital on July 2, 1924, as an emergency, having been involved in a machinery accident. Both hands had been crushed in a rolling machine, and had been held for some minutes before the machinery could be stopped and his release effected. On admission to the ward he presented a moderate degree of shock, being pale and sweating, pulse 64. The wounds had been dressed temporarily in the Casualty Department. Under general anaesthesia three hours later the condition of affairs was found to be as follows:—

*Right Arm.*—A complete cuff of skin had been torn down from half way up the forearm. The hand was severely crushed: multiple fractures were present; the thumb was almost torn off, and was hanging by the extensor tendons. Skin was avulsed from both aspects of the hand, and the soft parts were heavily infected with dirty material.

*Left Arm.*—The left hand was quite cold up to the wrist, and white in colour. Skin and subcutaneous tissues were lifted off the palm and part way up the forearm. Soft parts were extruded from two large lacerated wounds, and the hand felt like a bag of bones. The whole of the left forearm was bluish-red in colour.



OPERATION.—After very careful consideration it was decided that both hands were too severely injured to be conserved, and amputation was done through the lower third of both forearms. Short antero-posterior flaps were cut and loosely sutured over the ends of the stumps. Flavine dressings were applied, and antitetanus serum, 1500 units, given subcutaneously.

SUBSEQUENT COURSE.—The patient's general condition remained good, and the right forearm stump healed without trouble. On the left side a discoloration appeared, and finally became established dry gangrene of the distal half of the stump (*Fig. 383*).

On July 16 re-amputation was performed. The knife was carried circularly round the limb at the line of demarcation, without any attempt to fashion flaps. Wet flavine dressings were applied. No further extension of gangrene occurred, and the end of the stump gradually became covered over with healthy granulation tissue.



FIG. 383.—Photograph taken on July 16, 1924, showing gangrene of forearm stump prior to circular amputation.

#### PLASTIC OPERATIONS.—

1. On Aug. 18 a strip of skin and subcutaneous tissues measuring 6 in. by  $2\frac{1}{2}$  in. was lifted from the left side of the abdominal wall, and was resutured in position with its ends intact.

It was decided to perform the 'delayed flap' method in view of its effective result in limiting the blood-supply of the graft to its two extremities, as a preliminary to raising the graft from a single pedicle. The risk of gangrene, which might have occurred if such a long graft had been raised in one stage with its blood-supply dependent entirely upon vessels entering at one end, was thus avoided. At the same time it was a less cumbersome method than that of suturing the graft in the form of a tube, which always results in considerable shrinkage of the graft when the time comes for it to be unrolled.

2. On Aug. 29 the graft was raised again from the abdominal wall and freed at its outer end. The amputation stump was prepared for its reception by making a circular incision at the junction of skin and granulations, and obtaining a clear-cut edge of healthy skin to which the graft was to be sutured. The radius was shortened by half an inch. The pedicled graft was then swung across and sutured down over about three-quarters of the circumference of the stump. The arm was immobilized to the side in a

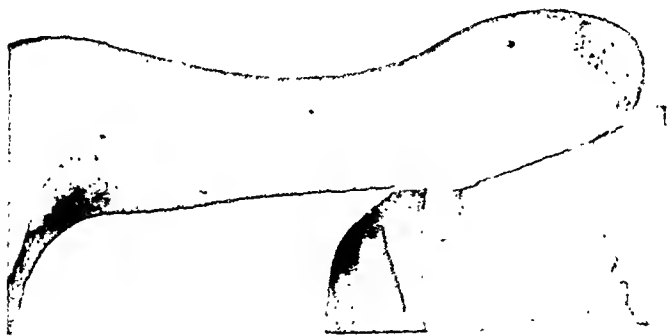
light plaster case. The bridging part of the graft was sutured to form a tube (*Fig. 384*).

3. Twelve days later, on Sept. 10, under local anaesthesia the graft was severed from the abdominal wall, and after being suitably trimmed it was



*FIG. 384.*—Circular amputation had been performed at the line of demarcation. The illustration shows the pedicled graft attached to the stump: Sept. 3, 1924.

sutured snugly down so as to cover the remainder of the raw surface entirely. For two days previously the attached end of the graft had been constricted for several hours a day by a rubber tube to encourage its new vascular



*FIG. 385.*—Lateral view of the stump: Nov. 7, 1924.

connection, and after its separation no anxiety was experienced in regard to its vitality. *Fig. 385* shows a lateral view of the stump on Nov. 7.

The patient was discharged on Oct. 3, 1924.

**RESULT.**—The result has been excellent. The end of the left forearm

has a smooth firm covering: the bones are not adherent, and in fact they have a better covering on the grafted side than on the opposite one. X rays show no sign of infection of the ends of the radius or ulna. There is a full degree of flexion and extension at the elbow, and a moderate amount of pronation and supination. There is a length of  $5\frac{1}{4}$  in. of ulna, which is an



FIG. 386.—Photograph of the patient fitted with his artificial arms, showing range of flexion at the left elbow: April, 1925.

ample length for an applanee. *Fig. 386* shows the patient with the artificial limbs which have been made by Messrs. Grossmith.

I am indebted to Miss Clephan for the series of photographs. My chief, Mr. A. E. Webb-Johnson, was kind enough to allow me to treat the patient throughout his stay in hospital. I am greatly indebted to him for his help and encouragement, and for his permission to record the case.

#### REFERENCE.

- <sup>1</sup> HUGGINS, G. M., *Amputation Stumps*, 1918, Oxford University Press.

## *SHORT NOTES OF RARE OR OBSCURE CASES.*

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### AN UNUSUAL COMPLICATION IN OPERATING ON LARGE TUMOURS OF THE NECK.

LIEUT.-COLONEL H. HALLILAY, I.M.S., CALCUTTA.

THE following case appears to be sufficiently uncommon to be worth recording.

S. K. B., age 20, Bengali, consulted me in June, 1925, about a large tumour on the right side of the neck which he said had been there since his early childhood. The tumour was a large lobulated mass occupying the whole of the space between the clavicle and the lower jaw on the right side of the neck. There was a striking under-development of the jaw on that side, and a condition of wry-neck towards the left. The right carotid could be traced from near the middle line along the inner aspect of the tumour to where it was lost behind the lower jaw. The larynx was displaced to the left of the middle line. The patient told me that he had been informed that any attempt to remove the tumour surgically would probably be fatal.

OPERATION, June 20, 1925.—Novocain and adrenalin. Transverse incision. It was necessary to tie off a large vein at the lower pole of the tumour, and one blade of a Mayo-Ochsner forceps was slipped under the vein preparatory to dividing it. As the blades were closed, the patient's voice died away into a husky whisper, and his pulse commenced to race. It being perfectly evident that in some way the fibres of the recurrent laryngeal had been caught up in the forceps, it was hastily disengaged, and the vein carefully dissected away from the capsule of the tumour. It was then seen that a nerve, suspected to be and subsequently identified as the vagus, was lying under and concealed by the vein. The nerve was stretched over the anterior surface of the tumour and was imbedded in the capsule, and it was necessary to dissect it out for the whole of its extent before the tumour could be released.

When the tumour was finally enucleated, the nerve was seen to be a much elongated vagus nerve, which, owing to the fashion in which it had been stretched by the growth, was about twice the normal length.

The remarkable feature of the case was the way in which the growth had separated the constituents of the carotid sheath, the right vagus being stretched over the front of the tumour, while the right carotid and jugular were displaced to the left of the middle line. The natural question which arises is, Where did the growth originate?

The tumour was examined by the Pathologist to the Medical College Hospital, Calcutta, Capt. Shanks, I.M.S., and was found to be a lipoma. It

weighed 40 oz. The patient made an excellent recovery, but had some degree of hoarseness and a rapid pulse for some weeks after the operation. The tumour had been diagnosed as a goitre, but the position of the carotid on the left of the tumour put this diagnosis out of court.

In conclusion, the incident affords a further testimony, if any were needed, of the virtues of a local anæsthetic in the removal of these formidable tumours of the neck. Had the operation been done under a general anæsthetic the vagus would inevitably have been divided—a misadventure which at the best would have led to the complete destruction of the patient's powers of phonation, and at the worst might have caused his death. Even the former would have been an irreparable disaster, for the patient intends to enter the Bar.

I have not encountered any mention of this complication in my reading, and would be much interested to learn if any one else has had a similar experience.

### SUPPURATING THYROGLOSSAL CYST.

By W. E. M. WARDILL, NEWCASTLE-UPON-TYNE.

THE following short note of a specimen in the Durham University College of Medicine Museum is offered as illustrating a cause of dyspnœa and dysphagia which was directly concerned with the death of the patient.

A female infant, age 3 months, was admitted to hospital in a state of collapse, with a history of difficulty in swallowing and breathing over a period of five weeks. The child was grossly emaciated, and there was marked indrawing of the intercostal spaces during each inspiration. A rounded, smooth, and elastic mass was palpable in the middle line of the neck immediately below the mandible.

At the autopsy, the specimen shown in *Fig. 387* was recovered. It consists of a thin-walled cyst, a little larger than a hazel-nut, which projects from the pharyngeal surface of the tongue and extends downwards as far as the base of the epiglottis. The median glosso-epiglottic ligament is stretched over the cyst, and is indicated by a slight longitudinal depression; both valleculæ are obliterated. The

cyst is filled with greenish pus, which, in the recent state, could be squeezed through the patent foramen cæcum. The inflammatory process does not extend to the surrounding parts: hence there is no œdema of the glottis. In front the cyst can be seen to form a small swelling limited below by the upper border of the hyoid bone. No abnormality was found in the lower part of the thyroglossal tract.

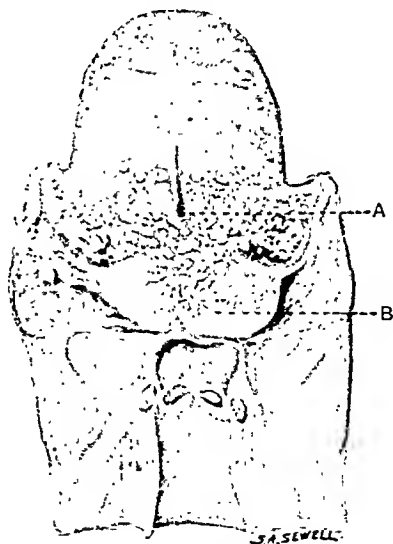


FIG. 387.—Thyroglossal cyst.  
A, Foramen cæcum; B, Cyst.

## MUCOUS CYST OF TONGUE.

By ERIC I. LLOYD, LONDON.

THE tongue here illustrated was removed post mortem from a girl of four months who was said to have suffered from impeded breathing for over eight weeks. Tracheotomy was performed, but the child died two days after admission to the Hospital for Sick Children, Great Ormond Street.

The illustration (*Fig. 388*) was drawn from the untouched specimen, and shows very well its general appearance. There is a cyst nearly half an inch in diameter in the mid-line of the dorsal aspect of the tongue half an inch behind the foramen cæcum. The cyst impinges against the front of the epiglottis, and by pushing it backwards partly closes the rima glottidis. The tumour is covered by normal mucous membrane, and the glosso-epiglottic fold runs along its superior surface.

*Fig. 389* shows the mounted museum specimen after hemisection of the tongue and removal of the roof of the cyst. The cyst was found to be thin-walled, and contained a semi-fluid substance which was not examined as the preparation had been long in a preservative solution. The inner surface of the cyst wall was smooth, and no channel or duct leading from it could be discovered. Serial sections were cut with the object of finding the remains of the thyroglossal duct, but no trace of this

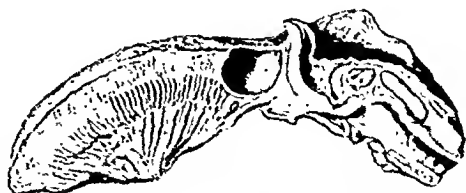
*FIG. 388.*—Showing size of cyst and position at base of tongue. (Natural size.)

was seen. The lining of the cyst was formed by a single layer of cubical cells, and external to this were alveoli and tubules of mucous glands. The thyroid and thymus glands were normal.

*Fig. 390* is drawn from a section, and shows the microscopic appearances.

The pathology of this rare congenital abnormality is obscure, but there are three possibilities :—

1. A dermoid cyst arising from an infolding of epiblast in the mid-line during development of the tongue is in some ways similar, but most of the recorded examples have been in the floor of the mouth, and such cysts usually have lining walls of thick epidermis with an appreciable amount of fibrous tissue; they may also contain hair and other epithelial products.



*FIG. 389.*—Hemisection of tongue, showing cyst with its roof removed. (No. 1162.2, R.C.S. Museum.)

2. A cyst developed in connection with the thyroglossal duct or its remnants may occur anywhere between the foramen cæcum and the isthmus of the thyroid gland; such a cyst is lined by ciliated columnar cells, and is most commonly found in the neck. The duct is normally replaced by a fibrous cord, but is lined, when it remains patent, by squamous epithelium for a few millimetres at its proximal (buccal) end, and distally by ciliated columnar epithelium. Probably in the tongue (1) and (2) are respectively buccal and pharyngeal variations of the same process occurring in different parts of the thyroglossal duct.

3. A mucous cyst may occur beneath the mucous membrane of the posterior third of the tongue where small racemose glands are normally found.

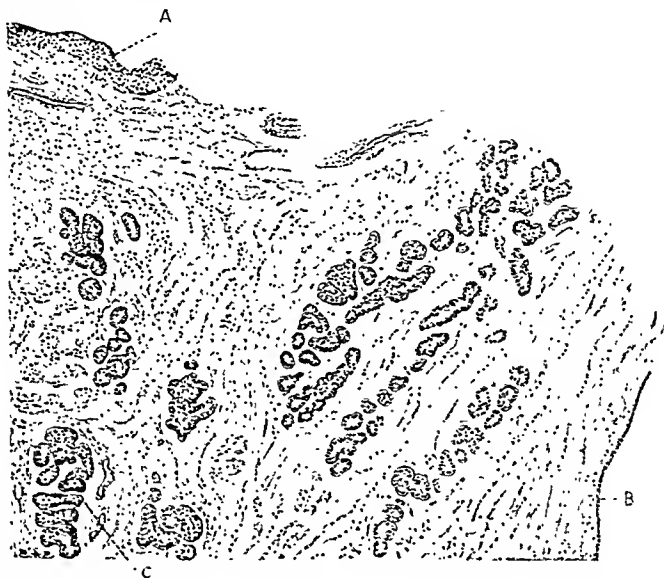


FIG. 390.—Microscopic section through tongue and wall of cyst. A, Mucous membrane of tongue; B, Cyst wall; C, Mucous glands. ( $\times 50$ .)

Such cysts are presumably due to blocking and subsequent distention of the ducts or alveoli; they are thin-walled, and seldom larger than a pea.

There seems no reason for associating this particular cyst with the thyroglossal duct, and from its position and its naked-eye and microscopic appearances, it must be classed as a mucous cyst.

The Museum of the Royal College of Surgeons of England possessed no example of this condition until Mr. George E. Waugh presented this specimen (1162.2). I am indebted to him for permission to describe what is both a clinical and pathological curiosity, and to Professor Sir Arthur Keith for his help and interest.

## RHABDOMYOMA OF THE TONGUE.

By GEOFFREY KEYNES, LONDON.

TUMOURS composed of muscle-fibres are uncommon in any part of the body other than the uterus, and I am unable to discover a record of any case of rhabdomyoma of the tongue similar to the one here described.

The patient was a traveller, age 26. For about six to nine months he had noticed a small white spot on the dorsum of his tongue. This had occasioned a good deal of irritation and discomfort, which was increased by smoking and had been made worse by the application of silver nitrate to the spot. He was first seen at St. Bartholomew's Hospital on Dec. 17, 1924, when he was found to have a small, hard, rounded lump, 0.5 cm. in diameter,

situated in the mid-line of the tongue half-way between the tip and the foramen cæcum. The lump was tender to pressure, but there was no appreciable change in the overlying epithelium, which was only slightly elevated above the surface, and there was no induration of surrounding tissues. A diagnosis of 'fibroma of the medium raphe of the tongue' was made, and the lump was excised under a local anæsthetic, together with a small wedge-shaped portion of the tissues around it.

No recurrence of the growth has since taken place, and nine months after the operation the tongue was found to be perfectly normal.

The cut surface of the tissue removed showed a




FIG. 391.—Low-power view ( $\times 45$ ) of part of the tumour with the overlying epithelium of the tongue. A bundle of normal muscle cells can be seen among the tumour cells on the right-hand side of the field.

rounded white area, apparently continuous with the overlying epithelium, and infiltrating the surrounding muscles. The microscopic appearances are seen in the accompanying figures. *Fig. 391* shows a low-power view of about half the tumour. The epithelium over it is quite unaffected, although it is in immediate contact with the tumour cells; these form a rounded mass of large, irregularly disposed cells, staining less intensely with eosin than the normal muscle cells, a few of which are interspersed among the tumour cells without being in any way altered. There is no trace of a capsule at the edge of the tumour, nor is there any sign of an inflammatory



reaction. In *Fig. 392* is seen a higher magnification of a group of tumour cells. These are large and elongated, with a very granular cytoplasm.



FIG. 392.—High-power view ( $\times 560$ ) of a group of tumour cells with associated endothelial and connective-tissue cells.



FIG. 393.—Oil-immersion view ( $\times 1040$ ) of a tumour cell lying between two unaltered striated muscle cells.

*Fig. 393* shows a still higher magnification of a tumour cell lying between two normal striated muscle cells. Their sizes and appearances may thus be

easily compared. No striation could be demonstrated in the tumour cells, even in sections stained with phosphotungstic acid; but a survey of the rather scanty literature of rhabdomyoma, such as is given in Ewing's *Pathology of Tumours*, shows that the cells are often found in an embryonal



FIG. 394.—Oil-immersion view ( $\times 1200$ ) of the cytoplasm of a tumour cell (right-hand side) and of an oblique section through a neighbouring striated muscle cell (left-hand side).

form without striation. In Fig. 394 are seen, side by side, very high magnifications of oblique sections through a striated muscle-fibre and through a tumour cell. The similarity is striking, though the organization of the tumour cell seems to stop just short of striation.

In spite of the absence of striation there can be little doubt of the nature of the tumour. If it is subjected merely to a process of elimination, it can only be identified as a rhabdomyoma. Sir Bernard Spilsbury has kindly examined the sections for me with the greatest care, and has confirmed this opinion. The tumour, though not encapsulated, seems to be innocent

in character, so that no treatment beyond the conservative operation already performed is likely to be needed in the present case.

The characters of the growth were scarcely well enough defined for a clinical identification of so rare a tumour to be made on a future occasion. The diagnosis must rest rather on the histological appearances.

The microphotographs illustrating this article have been made by Miss M. Vaughan, Dunn Laboratories, St. Bartholomew's Hospital.

## CYSTIC PNEUMATOSIS OF THE SMALL INTESTINE.

BY S. H. PUGH, TRAVANCORE, S. INDIA.

THE following case of cystic pneumatosis of the small intestine will be of interest, especially in view of the article by Cyril A. R. Nitch in the April, 1924, number of the JOURNAL.

Male Indian, age 39. Patient said he had vague symptoms of dyspepsia for the past fifteen years. There were, however, 'short intervals' when his digestion was good. For the last three years he had definite pain three to five hours after food, which was worse at night. He first noticed distention of the abdomen twenty months before admission. This became progressively

worse during the first ten months. During the second ten months the condition remained about the same. He vomited once or twice a month during the last three years, but never more frequently. The vomit rarely contained food, but usually consisted of 'bile or mucus'. He had long been losing weight.

ADMISSION.—March 29, 1924. The patient was emaciated, his weight being 78 lb. During the nine days he was kept under observation, his temperature was normal, and pulse varied from 68 to 84. There was slight distention of the abdomen. Constipation was not marked. There was no vomiting, but the appetite was poor.

OPERATION.—April 8. An exploratory laparotomy was performed through a mid-line incision above the umbilicus. Immediately the peritoneum had been incised, I was astonished by an audible escape of gas, and for an instant thought the intestine must have been opened. This was obviously not so, and the gas was odourless. That there had been no previous perforation of the stomach or intestine was also indicated by the absence of any sign of acute inflammation. The parietal peritoneum, and serous covering of the stomach, upper part of jejunum, and omentum were perfectly normal. There were no adhesions anywhere, and no free fluid in the abdominal cavity. No lesion was found in the stomach or duodenum. The small intestine, however, presented a remarkable appearance. From a point about three or four feet from the duodenojejunal flexure, on the surface of several sections of the bowel from two or three inches up to about three feet in length there were numerous blebs of all sizes up to about one-third of an inch. The blebs obviously contained gas, for when they were pricked they immediately collapsed, and apparently contained no fluid. Many of the blebs were exceedingly thin and transparent like soap bubbles. It is difficult to believe that these delicate blebs could remain long without breaking and liberating their contained gas. This would indicate that the gas was being formed at a considerable rate. Several of the affected sections of the bowel were greatly and irregularly thickened. On handling they were more rigid and heavier than normal. It was surprising that the action of the bowel had not been more interfered with. The increased thickness of the bowel suggested that there had been a long-continued but very mild irritation. There was no sign of congestion or indication that there had ever been any acute inflammation. The last three inches of the ileum and the whole of the large intestine appeared to be unaffected. So much of the bowel was involved that resection was impossible, and the abdomen was closed. The patient recovered from the operation, and his condition, when he left the hospital three weeks later, was much the same as when he was admitted.

This case is apparently the first of the kind reported in India.

Niteh draws attention to the association of gastric or duodenal ulcer in 50 per cent of the recorded cases. Duodenal ulcer is exceedingly common in Travancore. During 1924 in the Neyyoor Hospital the operation of posterior gastro-enterostomy was performed for that condition in 129 cases. While, therefore, one is familiar clinically with the condition and with the appearances at operation, in this case neither the symptoms nor the findings at the operation suggested gastric or duodenal ulcer. It seems unlikely, therefore, that the gas under pressure, such as that derived from aerophagy, etc., was

forced through an abrasion in the stomach or duodenum, especially in view of the fact that the ileum was more affected than the jejunum. It is, however, certain that the disease caused some, if not very much, obstruction in the small intestine. Yet it is difficult to believe that the obstructed gases were forced through any lesion in the intestinal mucosa. That a very large volume of gas was formed, and that its rate of formation was rapid, was indicated by the fact that the gas was being produced as fast as it could be absorbed even under considerable pressure by the serous surfaces in the abdomen. Would it be possible for so much foul air to escape from the bowel into the tissues without bringing acute inflammatory consequences in its train, and for the gas itself to become quite odourless, when it had reached the abdominal cavity?

Then the marked local thickening of the intestine, and the rather long history of the case, indicate that the irritating influence remained confined to the particular affected parts for a considerable period of time. It seems hardly likely that the same channel or channels for the escaping gas would remain patent for any great length of time, and so open into the same tissues.

On the mechanical theory it is evident that each bleb must have its own separate ultimate channel from which it receives its gas. That would necessitate many hundreds of channels, derived perhaps by branching from the main channel, sufficiently small to pass the gas slowly enough to give time for the formation of the blebs and to allow them to become of large size before they burst. It seems unlikely that the mechanical conditions could be so exactly reproduced in so many and widely separated parts of the bowel as to bring about the widespread similar thickenings of the bowel wall, and a great number of blebs of just the same appearance. This similarity of effect seems to suggest that the affected tissues were almost equally permeated with the cause of the condition. This is more suggestive of a chemical or bacterial agent than of a mechanical explanation.

On the other hand, if the mechanical explanation is not the true one, it is clear that the agent which produces the gas so rapidly must be a very active one. If the cause is bacterial, it is difficult to reconcile such great bacterial activity with the complete absence of all signs of acute inflammation. Moreover, if the presence of 15 to 20 per cent oxygen in the gas is confirmed in other cases than those of Krummacker and Urban quoted by Nitch, that would involve the difficulty that the microbe must be an oxygen-former. These two serious difficulties in the bacterial theory would seem to make the mechanical theory the least improbable explanation so far offered, in spite of the difficulties associated with that theory.

Since writing the above I have heard from the patient's friends, twelve months after the operation, that he improved a good deal soon after leaving the hospital. Practically all the symptoms disappeared, and he was able to enjoy ordinary food until three and a half months ago, when the old symptoms began to recur "with vomiting and eructations of gas [this may suggest aerophagy, which is not uncommon in Travancore], the vomiting becoming gradually worse, though there was some abatement for a short time, and he has now become so ill that he cannot leave his bed without help". His friends promise to bring him again to the hospital if he improves sufficiently, when further observation should be of interest.

## A CASE OF IMPERFORATE ANUS WITH MEGALOCOLON AND TERMINAL PERITONITIS.

By R. WATSON JONES, LIVERPOOL.

THERE are many features of interest in the case of M. L., female, age  $3\frac{1}{2}$ , who came under treatment at the Royal Infirmary in the early months of this year. The mother's complaint was that the child had no anus, and suffered from an incontinent discharge of fæces from the vagina. A history of constipation was elicited, with occasional attacks of diarrhœa, at which intervals the motion was inclined to be foul and offensive. Otherwise there were no symptoms, and the mother's worry was mainly a cosmetic one.

**EXAMINATION.**—An examination revealed the presence of an anal dimple but with no communication with the rectum. Vaginal inspection showed an opening in the posterior wall, at the junction of the middle and lower thirds, through which fæces were discharged. Some stress must be laid on the fact that the opening was not in the vulva, but well up in the vaginal wall above the hymen, which was present. The vagina was not septate and appeared otherwise normally developed.

**OPERATION.**—This was undertaken by Mr. Thelwall-Thomas, and as a first stage the rectum was brought down by perineal dissection to the position of the normal anus, opened, and stitched there. The wound healed rapidly, and the child was discharged. Two months later she was re-admitted, and an attempt was made to close the vaginal fistula from which fæces were still discharged in spite of the fact that the new anus was functioning. Convalescence progressed satisfactorily for three weeks after the second operation, when the patient suddenly became ill and vomited persistently. The abdomen was rigid and distended, and showed reversed respiratory rhythm. Laparotomy confirmed the diagnosis of general peritonitis and showed a markedly dilated and hypertrophied colon. On the third day the patient died.

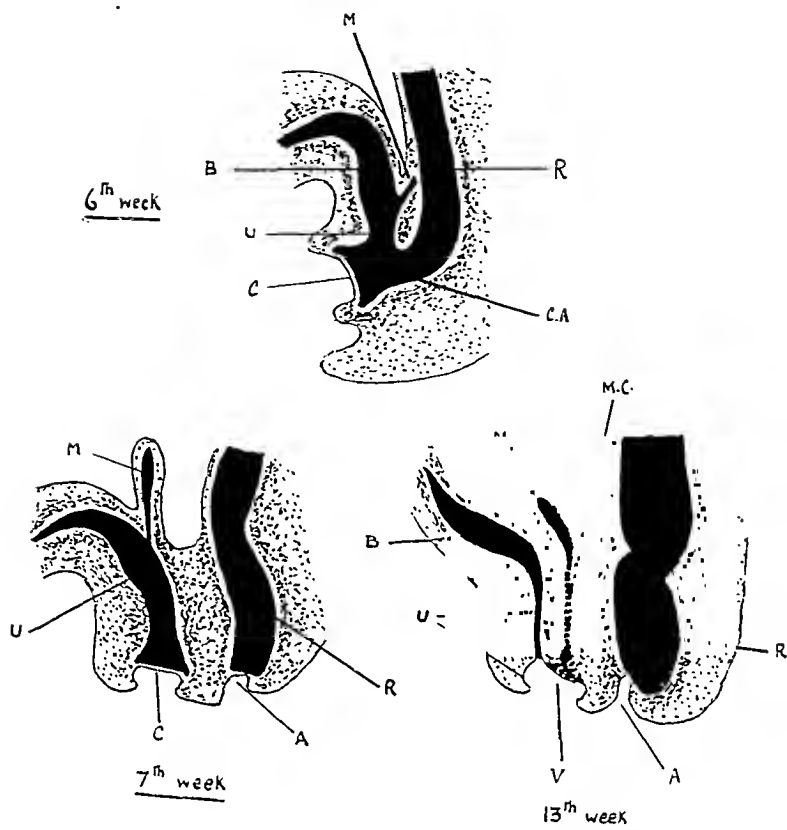
The case is of interest in the sudden onset of the final stage without any obvious direct cause. The patient had lived three and a half years without developing any signs of chronic obstruction with the sole exception of constipation and occasional diarrhœa. She was a fairly healthy and well-nourished baby; there had been no vomiting, no abdominal pain, no passage of mucus or blood, no abdominal distention, and there was no visible peristalsis. Presumably the vaginal opening was adequate, and certainly after operation the new anus was as efficient as, if not more efficient than, the previous orifice—its efficiency being ensured by the daily passage of a large-sized rectal tube, and the daily administration of an enema. Actually, however, there was a widely dilated and hypertrophied colon, containing large scybalous masses, though the dilatation was limited to the iliac and pelvic portions of the large intestine, the rest of the alimentary tract being normal. Although the probabilities are in favour of the condition being in reality that of chronic intestinal obstruction, the resemblance to congenital megalocolon (Hirschsprung's

disease) is marked. The definite hypertrophy shows that the condition was not of recent development, and the fact that the enlargement was strictly limited to the sigmoid colon and rectum is suggestive. It seems unlikely that the final events were ushered in as a result of the obstruction becoming more complete: indeed, the reverse seems to be the case; moreover, the clinical and post-mortem findings were those of a primary serofibrinous peritonitis, not of an acute intestinal obstruction. One is therefore led to the conclusion that two operations and hospital life had reduced the resistance of the child to the abnormal bacterial decomposition that had always been going on, so that the mucous membrane and walls of the colon had been invaded and a general peritonitis produced.

Another interesting anomaly in this case was revealed at autopsy, illustrating the truth of the statement that congenital abnormalities are frequently multiple. A prolonged search failed to discover the presence of a gall-bladder, the common bile-duct leading directly from the hepatic ducts. Section of the liver proved that there was no gall-bladder hidden by a complete *pons hepatis*, and there was no sign of the organ in the gastrohepatic omentum or elsewhere.

The case, moreover, is instructive from the embryological standpoint. It is a not uncommon combination to find with an imperforate anus the rectum opening into the vulva immediately posterior to the hymen. This corresponds with the most frequently found condition in males, where the rectal orifice is in the verumontanum of the prostatic urethra. It is rare, however, for the communication to be high up in the posterior vaginal wall. Keith<sup>1</sup> has described such cases, and states that in his experience they are associated with a septate vagina—presumably the presence of the rectum having interfered with the normal process of fusion of the Müllerian cords. In this case the opening was at the junction of the middle and lower thirds of the vagina. Such a condition cannot be accounted for by the generally accepted explanation of its development—namely, that first given by Wood Jones.<sup>2</sup> The theory is made clear by the diagrams below taken from Wood Jones's thesis (*Figs. 395-397*).

In the seventh week of development, the rectum migrates backwards, its cloacal orifice disappears, and the urogenital sinus is left with the Müllerian ducts opening into it. The vagina, however, is developed from a completely new structure, a down-growth of solid columns of mesodermal cells from the lower ends of the Müllerian ducts. Wood Jones believes that the whole of the vagina is formed in this way, by a canalization of these Müllerian cords. This will explain the condition where the rectum opens into the vulval cleft, for the vulva is certainly developed from the urogenital sinus. But how comes the rectum to communicate with the vagina if this be formed entirely from the Müllerian cords which are a later development, and if the urogenital sinus produces nothing more than the base of the bladder, the urethra, and vulva? The only satisfactory explanation is that in some cases at least, as suggested by Blair Bell,<sup>3</sup> the lower third of the vagina is developed from the posterior part of the urogenital sinus, cut off from the anterior part by the vesicovaginal septum. The Müllerian cords perforate the summit of this portion and form the upper two-thirds of the vagina (*Fig. 398*).



FIGS. 395, 396, 397.—Diagrams to show development of vagina according to Wood Jones. B, Bladder; U, Urogenital sinus; M, Müllerian ducts (uterus); R, Rectum; C.A., Cloacal anus; C, Cloacal membrane; A, Anus; M.C., Müllerian cords; V, Vulva.

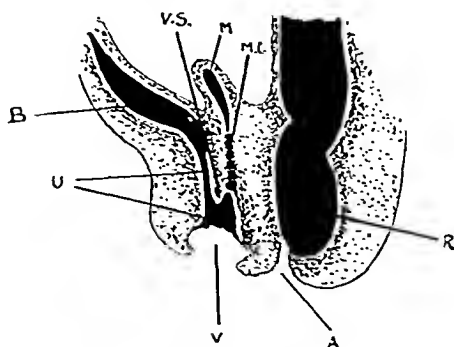


FIG. 398.—Diagram to show development of lower part of vagina from urogenital sinus—which permits of a vaginal opening of the rectum, being explained as a persistent cloacal anus. B, Bladder; U, Urogenital sinus; M, Uterus; M.C., Müllerian cords; V.S., Vesico-vaginal septum; V, Vulva; A, Anus; R, Rectum.

Such must be the explanation of the development in the case described, even if it is not true of all cases. The fact that the level at which the rectum may open into the vagina varies only illustrates the fact that varying proportions of the urogenital sinus may be included in the vagina in its development. Moreover the hymen cannot consist of the fringe left after the Müllerian cords have broken through on to the surface,<sup>5</sup>—that is, at the junction of the vagina with the urogenital sinus—but must develop in association with the breaking down of the cloacal membrane or urogenital plate, which originally closes the sinus below.

It is a pleasure to acknowledge my indebtedness to my chief, Mr. Thelwall-Thomas, for permission to investigate the case at autopsy and to publish the findings.

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- <sup>1</sup> KEITH, "Malformations of the Hind End of the Body", *Brit. Med. Jour.*, 1908.
- <sup>2</sup> WOOD JONES, "The Nature of the Malformations of the Rectum and Urogenital Passages", *Brit. Med. Jour.*, 1904, Dec. 17.
- <sup>3</sup> BLAIR BELL, *The Principles of Gynaecology*.
- <sup>4</sup> BERRY HART, *Edin. Med. Jour.*, 1911, vi, 577.
- <sup>5</sup> KEITH, *Human Embryology and Morphology*, 4th ed., 377.

## PULSATING GOITRE ACCOMPANIED BY RECURRENT DISLOCATION OF THE EYEBALLS.

By HAROLD BURROWS, PORTSMOUTH.

DISLOCATION of the eyeball, though it has been known to happen in little Pekinese dogs, and perhaps in some other animals, seems to have been a rare occurrence in mankind. Gradual forward displacement of the globe by an orbital tumour is, of course, a recognized condition; but the case about to be described is of another kind.

The patient, now aged 45, had been invalided out of the Police Force in 1918 after sixteen years' service, owing to a gradually increasing weakness which prevented him from getting through his day's round of a large rural district which included a military camp. He first consulted me in August, 1923, on account of repeated dislocation of the eyeballs, stating that any effort such as sneezing or yawning, or merely looking downwards suddenly to the ground, would cause his eyeballs to come out of their sockets unless he put up his hand to keep them in place. For the same purpose he used to throw his head backward, and these two precautionary movements had become habitual with him. The man himself was emaciated, and suffered from pulsating goitre, over which a loud bruit could be heard. This bruit was audible over the front of the chest as low down as the fifth costal cartilage in the nipple line on each side, and in both right and left inter-scapular regions; it was conducted also on to the cranium. Exophthalmos.



though pronounced, was not extreme (*Fig. 399, a*). Tachycardia was present in a moderate degree, the pulse-rate when he was at rest being usually at about 92 or 96. His weight was 8 st. 12 lb. He suffered from the usual nervousness and tremors of Graves' disease.

The most interesting feature of the case was the ease with which the eyeballs could be dislocated by digital pressure on the upper and lower lids (*Fig. 399, b*). When a dislocation had been produced in this way, the patient at once effected reduction by placing the pulp of his forefinger on the cornea and pressing the eyeball back into its proper position. It was manifest that the dislocation of the eyeballs caused him some momentary

*a**b*

FIG. 399.—*a*, Shows moderate exophthalmos and wide ocular clefts; *b*, Shows the left eyeball dislocated through the lids.

uneasiness and apprehension, and he lost no time in effecting reduction in the manner just described. His eyesight was unimpaired.

Partial thyroidectomy was performed on Nov. 15, 1923, the eyelids being stitched together in order to prevent the occurrence of a post-operative dislocation. These stitches, which caused a good deal of discomfort, were removed on the following day.

When seen in April, 1925, the patient's weight was 11 st. 6 lb., tachycardia had disappeared, and he said he could do anything. No dislocation of the eyeballs had taken place since the operation, although some degree of exophthalmos was still to be observed.

**THYROID GLAND TISSUE IN DERMOID CYST OF OVARY.**

BY ERNEST H. SHAW, LONDON.

THE following case of dermoid ovarian cyst is recorded on account of its unusual contents. As far as the writer can remember, it is the first specimen in which he has discovered thyroid tissue in a dermoid. The microscopic examination of teratomata is always interesting, and the varied picture seen in many of them affords the morbid histologist great pleasure. It seems so odd and weird to see bits of the various organs and tissues of the body jumbled together in one section—skin with its various appendages of sebaceous glands and hairs, mucous membrane with nice columnar-celled glands, striped and unstriped muscle, fat and fibrous tissue, etc. Even an incomplete ring of choroid with its pigments was found in one specimen.

A small portion of the specimen was received for examination and report from Dr. H. F. Ealand, who has kindly given permission for publication. Mr. Eric Sheaf, who removed the cyst, has been good enough to supply the clinical and other details, and the writer wishes to thank these gentlemen for their kindness.

The patient, Mrs. D., age 71, nullipara, had noticed a gradual and painless enlargement of the abdomen for some months. This had caused no inconvenience except some fullness. No difficulty of micturition or defecation. There had been no loss of weight or general health. She was found to be a spare healthy old woman, with a tumour in the abdomen, rising out of the pelvis and extending above the umbilicus. It was smooth, and movable laterally as far as its size permitted, tensely elastic in consistence, and dull on percussion. No dullness in the flanks or other abnormality was made out. No vaginal examination was made on account of difficulty caused by the hymen being intact. Diagnosis: 'Probably ovarian cyst'.

OPERATION.—July 31, 1925. A left paramedial incision exposed a bluish cystic swelling. On delivery its pedicle was found to be connected with the left broad ligament. The pedicle was tied, and the tumour removed in the usual way. The uterus and right ovary were normal except for a few very small fibroids in the uterus.

**MACROSCOPICAL EXAMINATION** of the cyst after removal showed it to be composed of several loculi, some containing thin fluid, one near the base with thicker wall containing the white thick oily material and hair characteristic of dermoids. On one side of the wall was a solid mass composed partly of cartilage, from its hardness ossified in places, and some soft tissue not unlike that of thyroid gland.

**MICROSCOPIC EXAMINATION.**—Layer of skin, thrown into folds, with a large number of sebaceous glands. Two hair roots are seen cut obliquely across. A few sweat glands are present in the underlying fat. A small patch of ill-formed bone is seen at one end of the section, and below this, and separated from it by a band of fibrous tissue, there is a large patch of thyroid tissue. Most of the thyroid vesicles are large and filled with colloid material. In some places the vesicles are quite small. (*Figs. 400 and 401.*)

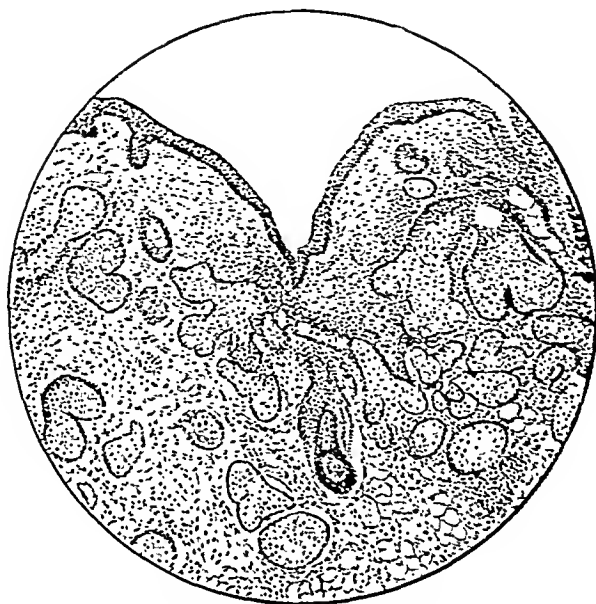


FIG. 400.—Illustrates the skin elements, with the large number of sebaceous glands which are so common in dermoids.

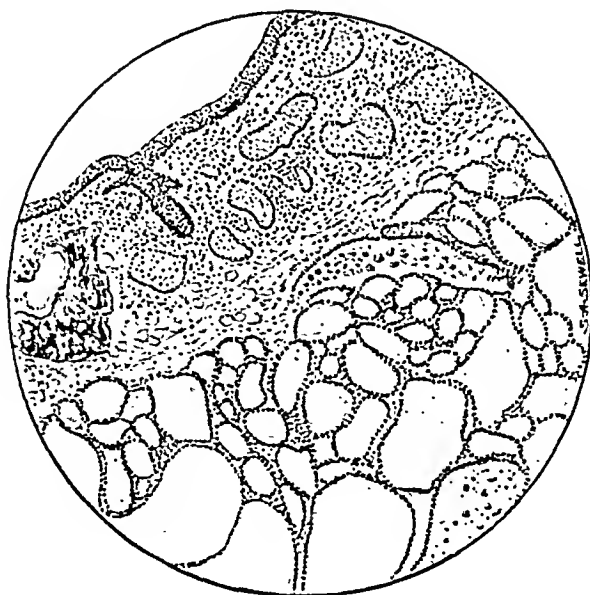


FIG. 401.—Also shows skin structures, with a dark patch of ill-formed bone on the left. Below this is a mass of typical thyroid vesicles. Two collections of brown-coloured cells indicate old blood extravasation. This is interesting in view of the fact that hæmorrhage is so common in thyroid adenomata.

duct was everted, and there was some organization of the surrounding blood-clot. It was through this that the bile was escaping into the peritoneal cavity, where it was absorbed into the blood-stream.

**Comment.**—There are very few cases of rupture of the hepatic ducts reported in the literature; since 1900 we can find only 15 recorded cases of traumatic rupture. Garrett reports a very interesting one in 1900. A farmer of 21, crushed between timber, was severely shocked, but after this passed off was comparatively well for ten days, when he became jaundiced and distended. He was aspirated twice, and in all 16 quarts were withdrawn. Laparotomy was performed on the twentieth day, and a rupture of the proximal part of the common bile-duct found. Drainage was provided and complete recovery followed. He also quotes a case of Spencer's, in which a child run over by a cab became jaundiced on the third day, and at operation  $1\frac{1}{2}$  pints of bile-stained fluid were withdrawn and the abdomen was drained. The abdomen was re-opened on the eighteenth day for a collection of fluid within the peritoneum. Death occurred on the thirty-third day, and post mortem the gall-bladder was found torn off. Porter reports a case which died forty-eight days after operation, with large sub-diaphragmatic abscess, and the common bile-duct was found completely severed. Hildebrandt mentions two cases in 1906. One, a child of 5, received a crushing injury; laparotomy showed rupture of the hepatic duct, and with drainage recovery was complete in four weeks. Waugh recorded an interesting case in 1915 in the *BRITISH JOURNAL OF SURGERY*. A boy of 6 was run over by the wheel of a car. There were no symptoms for three days, when he became jaundiced and distended, and bile was found in the urine and clay-coloured stools were present. At laparotomy  $1\frac{1}{2}$  pints of bile and blood were found in the peritoneum, and a tear of the anterior layer of the gastrohepatic omentum, the portal vein being plainly seen in this, but the common bile-duct was not seen. Drainage was provided, but seven days later the patient was jaundiced and distended, and the abdomen was re-opened and 2 pints of bile-stained fluid found in the lesser sac. This was repeated a month after the first operation, and recovery was complete in the end.

In studying these cases one is struck with the fact that they all have several important points in common. Following the severe collapse resulting from the injury there is a variable period of comfort and freedom from symptoms. Jaundice is the first sign to appear—in our case before the end of 24 hours; in Garrett's case it did not appear till the tenth day; the average is about three days (Desrosier). The reason for the period of quiescence is that normal bile is only slightly infectious and causes little peritonitis, except in cases of gall-stones, where a fatal peritonitis may be quickly set up (Keen). Later on more serious symptoms arise, such as increase of pain and tenderness, rigidity, distention, and vomiting. The absorption of bile into the blood-stream by way of the peritoneum has made the patient toxic and has damaged especially the heart muscle—probably by direct action of bile salts on the cardiac muscle—and the patient is now a very poor operative risk.

The recorded cases show that some recover when operated on some time after the accident (Garrett's case was operated on twenty days after), but the prognosis is shown to be fairly good if operation is done shortly after the injury, when the only signs present are the jaundice and some tenderness and rigidity in the region of the liver. Meyer states that 4.75 per cent of ruptured livers go into jaundice; but even if at operation only a rupture of the liver is found, the drainage of the peritoneal cavity is a beneficial thing, and at the same time the rupture could be attended to. When rupture of the bile-duct is found, repair, if possible, should be attempted; but if this is impossible, or the tear cannot be found, drainage is sufficient, as the rupture heals in time.

We are indebted to Mr. C. H. Shorney Webb for permission to publish this case, and for his kind advice and assistance.

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### HERNIA THROUGH THE FORAMEN OF WINSLOW: SECONDARY RUPTURE THROUGH THE GASTRO-HEPATIC OMENTUM.

By E. ROCK CARLING, LONDON, AND A. TENNYSON SMITH,  
ORPINGTON.

ONE of us (A. T. S.) was called to see a man, A. M., age 27, at midnight on Aug. 2, 1925, when he was complaining of severe pain in the upper abdomen. The day before he felt perfectly well; for supper he had a surfeit of dripping; on the morning of the 2nd he was sick, and vomited several times during the day. At 8 p.m. he was seized suddenly with severe pain in the abdomen; it persisted without relief.

When seen at midnight he was sitting up in bed, unable to lie down, and had recurrent paroxysms of colic. He had not the facies of the acute abdomen. Pulse 68, temperature 97°. He referred the pain to the upper zone of the abdomen, and said that it was worse when he tried to take a deep breath. His bowels had been open that afternoon. The abdomen was slightly distended in the upper half; there was tenderness in the epigastric region, but to no marked extent, and the muscles above the umbilicus were slightly rigid; they relaxed, however, when one engaged him in conversation till the next paroxysm of 'bursting pain' came on. There was no tenderness in the lower abdomen. Liver dullness normal. He could not sit forward for examination of his back, but rolled over in a three-quarter prone position. There was a tympanic note in the right posterior dorsal region extending up almost to the angle of the scapula, and his breath sounds were more distant than on the left side. He seemed to find relief from the semi-prone position; the pain left him, but returned when he resumed his former posture.

He was sick again during the night. He was admitted to the Cray Valley Hospital on the morning of the 3rd; by this time, though the local

tenderness was unaltered, the whole of the abdominal muscles were on guard and rather masked the distention noted the night before.

We saw the patient together at 10 o'clock on the morning of Aug. 3. He was half-sitting in bed, rocking himself to and fro in unbearable pain; it seemed located mainly in the upper zone of the abdomen, which was extremely rigid. There was but slight distention and no visible swelling; there was hyper-resonance over the liver area. It was doubtful if there was even the narrowest band of hepatic dullness just below the chest resonance, and in view of the fact that there was no general distention, this sign was interpreted as due to free gas, and a provisional diagnosis of leaking gastric ulcer was advanced.

On opening the abdomen, free fluid, slightly blood-stained but not turbid, was found; the narrow stomach lay noticeably low, and was reddened and distended; the small intestine immediately came into view above the lesser curvature. The loop came from behind through a gap in the gastro-hepatic omentum. Attention was at once directed to the foramen of Winslow, whence a much distended, and a collapsed, coil were found issuing. By combined pressure through the lesser sac and traction from the foramen the herniated gut was extracted. Though there were petechial hæmorrhages on the surface of the coil and on the mesentery, the bowel was viable, and was returned to the general abdominal cavity over the front of the transverse colon. The rent in the lesser omentum, which appeared to be quite recent, was repaired, but œdema around the foramen was such as to make suture very hazardous, whilst the abdominal wall was rigid and straining, and it was not attempted.

Before the abdomen was closed, the parts had resumed a normal arrangement and the only thing noticed about the transverse colon was that the great omentum was slight, scanty, and fat-free. The coil herniated was ileum rather above the mid-point of its extent, and its mesentery seemed to be a little unduly long, with perhaps rather a restricted attachment area on the posterior wall; but examination was certainly difficult. The length of bowel in the lesser sac was about 20 inches.

Post-operative recovery was uninterrupted; the man left hospital after a fortnight, and has since returned to his work.

Ullman,<sup>1</sup> in recording a case of his own, has recently given abstracts of twenty-nine others; he includes eight not mentioned in Rendle Short's<sup>2</sup> list, but omits two or three there alluded to; finally, there is McKenzie and Wood's<sup>3</sup> case, the figure from which, with slight modification, would do very well for ours. Amongst all the cases there is only one in which secondary rupture through the lesser omentum occurred, that of Schmiliusky.<sup>4</sup> In Blandin's<sup>5</sup> case the herniated gut re-entered the greater sac through the transverse mesocolon.

#### REFERENCES.

<sup>1</sup> ULLMAN, *Surg. Gynecol. and Obst.*, 1924, xxxviii, 225.

<sup>2</sup> SHORT, A. RENDLE, *Brit. Jour. Surg.*, 1925, xii, 456.

<sup>3</sup> MCKENZIE and WOOD, *Ibid.*, 613.

<sup>4</sup> SCHMILINSKY, *Deut. med. Woch.*, 1919, xlv, 477.

<sup>5</sup> BLANDIN, 1824, *Traité d'Anatomie topographique*, 467.

## ACUTE ASCENDING PARENCHYMATOUS ENTERITIS CAUSING PARALYTIC ILEUS.

By ZACHARY COPE, LONDON.

ANY light which can be thrown on the question of paralytic ileus should be welcome, so that the recounting of the following case may prove of interest :—

F. B., a man, age 28, was taken with acute abdominal symptoms on March 18, 1924. When first I saw him on March 21, he was clearly suffering from acute appendicitis. At the Bolingbroke Hospital on that date I removed a very acutely inflamed and gangrenous appendix which lay against the right wall of the pelvis, under cover of a hood of inflamed omentum. The contaminated omentum was removed, together with the appendix, and drainage by rubber tube instituted. All went well for three days, but on March 24 the patient began to vomit copious quantities of fluid, which soon became fæulent. There was no pain, and the pulse remained at 88, but collapse soon supervened; the cheeks became sunken, the pupils dilated, and as soon as I saw him it was clear that there was no time to be lost before remedying the obstruction. On re-opening the wound the last coil of the ileum was found obstructed and adherent to the right wall of the pelvis. It appeared difficult to clear it, so enterostomy, with short-circuit of the base of the obstructed loop of gut, was carried out. Next morning the patient was much better, and the enterostomy had acted well, but later in the day the flow almost ceased. Vomiting re-started, and the patient again became collapsed. The abdomen was now opened higher up and enterostomy performed in a coil of jejunum. At this operation it was noted that the lower part of the small intestine was enormously thickened with inflammatory œdema, and an attempt was made to open the gut above the inflamed area. There was no evidence of peritonitis. In order to lessen the amount of fluid lost, the suggestion put forward by Wilkie was adopted, and the upper and lower enterostomies were connected by tubing which permitted the fluid escaping from the one opening to re-enter the bowel by the other. For three days the patient's condition improved, but on March 28 the upper tube ceased to drain well, and the patient vomited pints of bile and again looked almost moribund.

The upper wound was then re-opened, and it was found that the thickened œdematous condition had spread farther up the small intestine, beyond the enterostomy. The second enterostomy was therefore closed, and a third one made still higher up at a point where the jejunum was closely in contact with a perfectly healthy-looking transverse colon. It was specially noted that there was no peritonitis, and the contrast between the distended, very thickened jejunum and a contracted, normal-looking colon which was lying adjacent to it was very striking. To make drainage of the small bowel more complete, a portion of the upper jejunum was also anastomosed to the transverse colon. The stomach was also washed out.

At each of these three operations two pints of saline were given intravenously to combat the shock. At no period did the patient complain of

any pain. From that time improvement began, and after a prolonged convalescence, during which the enterostomies were closed, recovery took place. A recent X-ray photograph shows that there is no obstruction, though attacks of colicky pain arise occasionally.

The above case illustrates a condition which I do not think has hitherto been recognized or described. From observation of the gut at the different operations, it was clear that there was an ascending inflammation of the small bowel, starting from the primary site of obstruction, involving the whole thickness of the bowel wall, and gradually ascending almost to the duodeno-jejunal junction. There was no peritonitis, for the large bowel was normal. The absence of pain was presumably due to the paralysis of the nerves in the gut wall by the inflammatory process. The condition in no way resembled those cases of ascending peritonitis which are well known and which have been well described by Handley; it was clearly due to an enteric and not a peritoneal infection. As the state of inflammation extended higher up the gut, the intestinal paralysis reached one enterostomy after the other. The walls of the bowel as observed at the operation were in remarkable contrast to the condition seen in peritonitis. Instead of being thin and almost transparent, they were very thick and œdematous, but not so congested or inflamed in appearance as occurs in peritonitis with ileus.

The corollary might be added that it is sometimes unwise to remove protective omentum in cases of appendicitis if the risk of adhesions is thereby increased.

It is impossible to appraise correctly the influence that the jejunocolic anastomosis had in this case. It was carried out because the writer knew Mr. Handley had achieved success in ileus duplex by a somewhat similar anastomosis.

A drawback which may be attributable to this short-circuit is that the young man now has a portentous appetite, and the bowels are opened rather soon after a meal.

#### CONCLUSIONS.

1. A condition of acute ascending parenchymatous enteritis may follow mechanical obstruction of the lower end of the small intestine.

2. The inflammation involves all the coats of the bowel and causes paralytic ileus.

3. The condition is unaccompanied by pain, owing presumably to the toxic effect of the products of inflammation on the nerve-endings in the bowel wall.

4. For its cure a high enterostomy is needed.



## REVIEWS AND NOTICES OF BOOKS.

**Operative Surgery: Covering the Operative Technique involved in the Operations of General and Special Surgery.** By WARREN STONE BICKHAM, M.D., F.A.C.S., New York. Vol. VI and Index Volume; pp. 908, with 1224 illustrations. The whole work complete in six volumes, and free index volume. In complete sets only. London and Philadelphia: W. B. Saunders Co., Ltd. Per vol. 50s. net.

THE six volumes of this work on operative surgery are a testimony to the erudition, to the patient industry, and to the sound judgement of the author. Not only are the ordinary, and indeed many of the unusual, operations described, but there are chapters which may be regarded as forming almost special treatises dealing with the operations upon the pelvic viscera of the female; upon the eye: and upon the ear, nose, and throat. We can think of no work published in modern days which so completely covers so extensive a field. There are a thousand pages, on the average, to each volume, and there is one illustration to every page. Such a range of subjects is rarely attempted by a single author; we expect nowadays to find a multitude of writers each dealing with a subject he has made his own. Dr. Bickham's book avoids the discrepancies and the overlapping and the bare patches which every encyclopædia by many authors seems always to display. Yet it cannot be said, in fair criticism, that any part of the vast subject dealt with in the six volumes is inadequately considered.

In a preface that is perhaps overlong, the author's intentions are disclosed. Though he rightly places a high importance upon technique, upon craftsmanship, he recognizes that judgement in the proper selection of a procedure applicable to a particular case outweighs all other considerations. The number of alternative methods described is sometimes a little bewildering, and to the unpractised surgeon this is a disadvantage. No wise guidance is given as to the most apt procedure in such and such conditions, and the earnest student may rise from a careful reading of any chapter knowing exactly in what precise fashion many operations can be done, but undecided as to which one ought in any instance to be selected.

There are omissions here and there—strange omissions sometimes—and there are operations described which should find no place in any modern work. Examples of the former are found in the chapter dealing with thoracic surgery, and of the latter in connection with the surgery of the peripheral nerves.

No detailed description of this work is possible in limited space; but we can give a very cordial welcome to these volumes. They will be of the greatest help to every advanced student, and they may well find a place upon the shelves of the surgeon who needs from time to time to refer, especially for teaching purposes, to the approved methods of other surgeons. The illustrations and letterpress are excellent.

**The Early Diagnosis of the Acute Abdomen.** By ZACHARY COPE, B.A., M.D., M.S. Lond., F.R.C.S., Senior Surgeon to Out-patients, St. Mary's Hospital; Senior Surgeon to the Bellingbroke Hospital; late Hunterian Professor, and Arris and Gale Lecturer, Royal College of Surgeons. Third edition. Pp. 233 + xiv, illustrated. London: Oxford Medical Publications. 10s. 6d. net.

THE fact that this book has already reached its third edition is sufficient proof of its value as a guide to the early diagnosis of acute abdominal disease.

The section on acute pancreatitis has been entirely rewritten. The symptoms of this disease are divided and described under five headings: (1) Those due to

inflammatory tension in the gland; (2) Those due to swelling of the pancreas; (3) Those due to extravasation of blood; (4) Those due to deranged gland function; and (5) Other symptoms. The author emphasizes the fact that profound shock usually accompanies the pain, and that the pulse is rapid and weak. This immediate rapidity and weakness of the pulse is to our mind one of the most important signs of the disease, and serves as an important factor in the differential diagnosis between this condition and perforation of a gastric or duodenal ulcer, in which we maintain there is no immediate rapidity and weakness of the pulse. Amongst other symptoms mentioned are pain in the loins and cyanosis. In the large majority of cases the pain in the back is intolerable, and is one of the most prominent symptoms in this condition. Cyanosis of the face and extremities, and in many cases of the skin of the abdomen, occurs so frequently that it may almost be taken as a pathognomonic sign.

In the preface to the third edition the author mentions our criticism of his 'stage of primary shock' in perforation of a gastric or duodenal ulcer. It is very necessary to define what is meant by 'clinical shock' in cases of this kind. We believe it is incorrect to describe a patient as suffering from clinical shock in the absence of a rapid pulse and lowered blood-pressure. In the first and second editions of the work this stage was said to last for "an hour or two". In the third edition the author states that "it may last for but a few minutes or persist for an hour or two".

We think that this is an important alteration, as we believe that this stage, if it exists at all, only lasts for a few moments, and in no case have we seen it persist for an hour or two. It is important to emphasize this point, as, if the author's teaching be followed, many will look for the signs of clinical shock—that is, rapid pulse and lowered blood-pressure—in the early stage of perforation, and failing to find them may delay operation at this the most hopeful period.

The author quotes from Moynihan's book on duodenal ulcer thus: "When perforation occurs there is usually a sudden onset of the most intolerable agonizing pain. This pain is hardly exceeded in severity by any that a human being can suffer; the extremity of agony is reached. So profound may the instant impression be that death results. . . . The patient is always prostrate with agony, he looks pale and faint, his face wears a deeply anxious expression, the eyes are wide and watchful, beads of sweat stand out upon his brow, and lines are quickly graven on his cheek". This, he says, could hardly be bettered as a picture of clinical shock, if there be any significance in the word 'shock'. We believe there is some significance in the meaning of the word 'shock', and we believe it to involve, amongst other signs, a rapid pulse and a lowered blood-pressure, neither of which is mentioned in the above quotation. The whole point seems to be a definition of the word 'shock'. True, the patient may look 'shocked' in the social sense of the word, but from a medical point of view—and surely the work is written for the benefit of medical men—the two main signs of clinical shock are absent. We have never yet seen a case presenting the signs of clinical shock as we understand it within a few moments of perforation. We have taken the pulse-rate and the blood-pressure in a large number of these cases, but have failed to find either a rapid pulse or a lowered blood-pressure in the early stages of perforation. One case perforated in the X-ray room. He was in great pain and looked pale and ill, but his pulse-rate and blood-pressure, which were taken immediately, were normal.

We feel that it cannot be too strongly emphasized that patients in the early stage of a perforation do not have a rapid pulse or lowered blood-pressure. The pulse is one of the first things a practitioner observes in an acute abdominal catastrophe, and if he discovers it to be normal and does not realize that it usually is normal in the earliest stage in perforations, he may lose valuable time in opening the abdomen.

Apart from this one point we are in entire agreement with the author's views as set forth in this excellent and helpful little book. It will continue to enjoy the position it has gained, and we can strongly recommend it to all who are interested in this subject. It is undoubtedly the leading book of its kind on this subject to-day.

**Abdominal and Pelvic Surgery for Practitioners.** By RUTHERFORD MORISON, Hon. M.A. and D.C.L., Hon. LL.D., M.B., F.R.C.S. Ed. and Eng., Consulting Surgeon, Royal Infirmary and Dental Hospital, Newcastle-on-Tyne. Crown 8vo. Pp. 212 + x, with 9 line drawings. 1925. London: Oxford Medical Publishers. 8s. 6d. net.

This small book is intended chiefly as a guide to diagnosis, and not as a guide to treatment. The author is impressed with the fact that the diagnosis and treatment of serious disease is now left largely in the hands of specialists, and further that the patient only seeks the advice of such specialist under the guidance of uninformed instinct. He would reinstate the general practitioner into the important position which he held more than thirty years ago, when the family doctor was always the first to be consulted about illness, and made it his business to make a diagnosis and then to advise consultation with a specialist if necessary.

The matter and manner of the book are most excellent. Brevity, clearness, and simplicity mark every page, and illustrative examples are given of all the conditions described.

**The Radiological Examination of the Male Urethra.** By G. L. S. KOHNSTAM, M.R.C.S., and E. H. P. CAVE, M.B., B.S., etc., late of King's College Hospital. With a Preface by Sir JOHN THOMSON-WALKER, O.B.E., M.B., C.M., F.R.C.S. Pp. 116 + xvi, with 64 illustrations. 1925. London: Baillière, Tindall & Cox. 15s. net.

THE authors of this monograph have applied to the investigation of the male urethra the methods employed by Voelcker and Liechtenberg in the study of the ureter and the pelvis of the kidney. They have, however, abandoned the use of sodium bromide as too irritating, and have finally selected lipiodol, diluted with four times its volume of sterile paraffin. An excellent account is given of the technique of the injection and of the arrangement of the patient and the X-ray tube during the taking of the skiagrams; the latter detail is of importance owing to the difficulty of getting an unobstructed view of the deeper parts of the urethra. A description of the appearances presented by the normal urethra is followed by chapters on deviations from the normal as shown by 'urethrograms' of cases of stricture, of false passages, urethral calculi, of the prostate before and after operation, and of sphincteric inefficiency.

Amongst many interesting observations we would single out as of special interest: (1) Urethrograms showing that, after suprapubic prostatectomy, sphincteric control appears to be taken over by the remains of the prostatic urethra rather than by the compressor urethræ; Sir John Thomson-Walker, in his preface, is of opinion that further observations are necessary before accepting this rather startling innovation. (2) The frequency with which the injection flowed into the vesiculæ seminales. (3) The appearance of relaxation of the bladder neck, called 'funnelling' by the authors, which is found associated with some cases of growth of the bladder and with some spinal diseases; this might prove of real diagnostic importance in cases where cystoscopy is difficult.

There are numerous radiograms and some tracings; the latter are very much easier to understand than the former. The authors are to be congratulated on an excellent piece of pioneer work.

**Facial Surgery.** By H. P. PICKERILL, C.B.E., M.D., M.S., Surgeon-in-Charge, Facial and Jaw Department, Durnedin Hospital. With an Introduction by Sir W. ARBUTHNOT LANE, Bart., C.B., M.S. 4to. Pp. 162, with many illustrations, and frontispiece in colour. 1924. Edinburgh: E. & S. Livingstone. 21s. net.

This volume represents a very laudable effort to concentrate within the confines of a comparatively small book, at a reasonable price, the extensive experience of the author in plastic surgical methods. We hold the opinion that this task is an impossible one, for this branch of surgery has increased so rapidly in its scope and literature since the war, and necessarily requires such profuse illustration by diagrams and case records, that it would seem to us better that an author should confine himself to the treatment of some particular branch of the work in any single volume. A

for removal of cysts, with four figures (source not mentioned or acknowledged), but no mention of Handley's name or work.

The chapters on the thyroid, parathyroids, thymus, and adrenals are by Sudeck. They contain an excellent and well illustrated account of the histology of various thyroid affections and also of all the stages of exophthalmic goitre and its treatment. The special characters of metastasis in thyroid tumours are mentioned but very briefly, and are not illustrated at all. Kocher's work dominates this section, with notable omissions of some other authorities. Oehlecker deals with the surgery of the hypophysis, and gives a short but complete account of its anatomy, functions, and the methods of surgical approach. In the latter the transfrontal route is mentioned as the best, but it is the temporal route which is illustrated.

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**A Text-book of Operative Orthopedics.** By A. STEINDLER, M.D., F.A.C.S., Professor of Orthopedic Surgery, State University of Iowa. Royal 8vo. Pp. 403 + xv, illustrated. 1925. London: W. Appleton & Co. 30s.

THE author's name having become well known as the originator of a special operation for claw-foot, his book will attract considerable attention. In the early parts of the volume, especially the section on tendon transplantation, our expectations are fully realized; but in the later chapters, and notably in those dealing with reconstructive bone operations, we are disappointed. The aim and general design of the book are very good. The author sets out to examine, first, the rationale of each operation as judged by its physiological and mechanical basis, giving special weight to experimental evidence. The clinical aspect is mentioned very briefly. The description of the operative technique and steps of the operation naturally occupies the biggest share in the book, and this is illustrated freely by diagrams, which are clear though sometimes crude, and by reproductions from original papers. There is an attempt to give a statistical account of operation results. This is certainly an excellent idea, though it is, of course, open to all the fallacies which are inevitable in trying to express complicated ideas in terms of simple numbers.

Each chapter ends with a list of the chief papers and references, and this seems to be well chosen. It has no claim to completeness, but it does serve to help the reader to get in touch with the principal authorities on any subject.

The most serious criticism of the book is that it attempts to deal with far too much in a very short compass. If a book of this size had been devoted to what used to be called orthopaedics, i.e., the surgery of deformities, the subject could have been adequately treated. But subjects such as spinal-cord surgery, and the surgery of fractures and joints, are also included, and these sections would require a much larger space to do them justice. Again, in order to achieve completeness, methods of doubtful value, e.g., cinematization of stumps and ramisection, are described, but not with sufficient detail to serve as a useful guide. It is undoubtedly a good book, but in future editions we venture to suggest that the author will have to choose between dealing thoroughly with a small subject or else greatly enlarging the work.

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**The Surgery of Pulmonary Tuberculosis.** By JOHN ALEXANDER, M.A., B.S., M.D., Assistant Professor of Surgery in the Medical School, University of Michigan, etc.; with Introductions by HUGH CABOT, C.M.G., LL.D., M.D., F.A.C.S., Professor of Surgery and Dean of the Medical School, University of Michigan, and EDWARD R. BALDWIN, M.A., M.D. Medium 8vo. Pp. 356, with 53 engravings and 12 plates. 1925. London: Henry Kimpton. 21s. net.

THE bibliography at the end of Dr. John Alexander's book contains 500 references to contributions on the subject of the surgical treatment of pulmonary tuberculosis, for the seven years 1918 to 1925. This is a clear indication of the extensiveness of the work which has been done and of the interest which has developed in this subject in recent years. The perusal of such a wealth of literature presents a formidable task even to the man keenly interested in the subject; owing to time and language difficulties, it is an impossibility to many. Dr. Alexander has therefore rendered a very great service to physicians and surgeons by extracting from this literature

all the salient facts. He has, in connection with each mode of treatment, brought together in a concise form the views of all those who have had practical experience of the methods. Where there is divergence of opinion, the various arguments have been very fairly compared and judiciously summed up. For this reason the book is essentially one to be read and studied by the physician and the surgeon. The former will obtain from it an extremely fair knowledge of the immense value of surgery for pulmonary tuberculosis, but will at the same time learn the limitations. For the surgeon there is, in addition, a wealth of information as to the technique of the various operations, and variations of the same type of operation.

Having traced the evolution of thoracic surgery, and gone at length into the indications and contra-indications for surgical intervention, the author deals with all the modern operations: extrapleural paravertebral thoracoplasty, phrenicotomy (under which heading is included exaeresis or evulsion of the phrenic nerve), extra- and intrapleural pneumolysis, and drainage of cavities, tuberculous effusion, and empyema. Artificial pneumothorax is not included, but there is a chapter on the comparative values of artificial pneumothorax and thoracoplasty. This comparison is extremely fair, and is of great assistance in view especially of the increasing tendency on the part of some of the German surgeons to advocate thoracoplasty.

The author shows of what great value the various surgical measures have been in saving lives which would otherwise undoubtedly have been lost. He emphasizes, and rightly too, how erroneous is the general impression of thoracoplasty; that the 'hazy notion' of the terrific shock and mortality, of the pain and post-operative deformity, is in no way based upon fact (p. 19). He has collected a total of 1159 thoracoplastic operations. The percentage results are: Cured, 36.8; improved, 24.4; living but unimproved or worse, 5.25; dead from causes directly or indirectly connected with operation, 14.1; dead from causes not directly connected with operation, 33.5. It must be remembered, he points out, that all these were advanced cases and all of them had had (with few exceptions) a prolonged course of sanatorium treatment, and possibly artificial pneumothorax treatment, which had failed to arrest the disease.

Dr. Alexander points out what a very small number of those patients who are suitable are offered the chance of surgical treatment, and of the possibility therefore of 'cure' or improvement. The great object of his book is to bring to the notice of those who have to advise these sufferers from pulmonary tuberculosis the possibilities of happy intervention—possibilities which are at the present too generally neglected.

It is interesting to note that "this monograph has been awarded the 1925 quinquennial Samuel D. Gross prize by the Philadelphia Academy of Surgery".

**Selected Papers: Surgical and Pathological.** By F. T. PAUL, D.Sc. Liverp. (Hon. Caus.), Ch.M., F.R.C.S., Consulting Surgeon, Liverpool Royal Infirmary. Demy 8vo. Pp. 284, with 23 plates. 1925. London: Baillière, Tindall and Cox. 15s.

A COLLECTION of published papers by Paul, of Liverpool, comes to this Journal for the purpose of review; but it would seem that they should be rather the subject for appreciation than for a review, for they indicate the feeling of the surgeons of Liverpool to one of their oldest colleagues. No better compliment or mark of respect could be paid to a man than such a gift from his own associates.

Frank Thomas Paul was born on Dec. 3, 1851, at Pentney, Norfolk. He was educated at Yarmouth Grammar School and Guy's Hospital, where he gained an exhibition and held the office of house physician. He qualified in 1873, and in 1878 became a Fellow of the Royal College of Surgeons of England. He was appointed Surgeon to the Royal Infirmary, Liverpool, in 1890, and also held the office of Dean of the Medical Faculty and Professor of Medical Jurisprudence in the University of Liverpool. An all-round man, a careful observer, interested in many things, Paul has made valuable contributions on many topics of surgery and pathology, but his name will ever be associated with the surgery of the large bowel. Undoubtedly it is due to him that the surgery of the colon is what it is to-day, and all students are familiar with 'Paul's tubes'. We would like to associate ourselves with the Liverpool surgeons in our congratulations to Mr. Paul.

great many of the cases recorded were treated at the Queen's Hospital, Sidcup, where the author had charge of the New Zealand section. In a rapidly developing and comparatively new branch of surgery it is always difficult to decide to whom the credit of introducing some new procedure belongs: it is difficult to believe that the author was entirely responsible—working as he did in close association with the heads of the other sections of the hospital—for all he lays claim to. There is, however, no doubt that great credit is due to him for having modified and simplified the Esser principle of pressure skin-grafting.

The work is well arranged in three parts: principles, methods, and technique of plastic surgery; military facial surgery; facial surgery in civil practice. The illustrations are very well reproduced. We fail to see the use of that on p. 4, in which "one hundred and forty life-size wax casts of typical facial injuries" are depicted crowded within the dimensions of a single page.

We do not agree with the author in his condemnation of the method of treating gunshot wounds of the mandible by fixation of the fragments in correct relative position to the maxilla, and we do not clearly understand his use of the words in italics, 'accurate approximation'. We feel that it would be better to get non-union in correct position—treating the case later by bone-grafting—than to obtain union in malposition by approximating the fragments. No mention is made of the difficulty of retaining the edentulous short posterior fragment in good position during the process of repair.

We are of opinion that skull defects are much more easily and economically filled by celluloid plates than by grafts from the tibia. The latter invariably become fibrous, and frequently give considerable trouble by becoming adherent to the dura and brain scar. It is well known that a very large number of grafts of this type have called for removal for this reason, and in most of these cases celluloid plates have been substituted with success.

Concerning syphilis of the nose, no mention is made of the need for replacing the loss of mucous membrane—the probable cause of the bulk of the deformity—by skin-grafting before inserting a cartilage graft to restore the bridge line.

**A Text-Book of Surgical Pathology.** By C. JENNINGS MARSHALL, M.D., M.S., F.R.C.S., Assistant Surgeon to Charing Cross Hospital and to the Victoria Hospital for Children, and ALFRED PINEY, M.D., Ch.B., M.R.C.P., M.R.C.S., Director of the Institute of Pathology, Charing Cross Hospital. Demy 8vo. Pp. 469 + vii, with many illustrations. 1925. London: Edward Arnold & Co. 21s. net.

THIS is a student's text-book of surgical pathology, and will no doubt serve its purpose extremely well. There was need for a new work on this subject, and the collaboration of an active surgeon and working pathologist is calculated to make the book a success. The matter is well arranged, and the information supplied just what the student requires. Many of the illustrations are reproductions of photographs, and show so little that they might well have been left out. This is a pity, as good drawings of pathological specimens are easily obtained.

**Malignant Disease of the Testicle: its Pathology, Diagnosis, and Treatment.** By HAROLD R. DEW, M.B., B.S. Mel., F.R.C.S. Eng., F.A.C.S., Hon. Surgeon to Outpatients, Melbourne Hospital. Royal 8vo. Pp. 168, with 52 illustrations, including 5 plates in colour. 1925. London: H. K. Lewis & Co. Ltd. 21s. net.

THIS valuable monograph embodies the chief facts put forward in the author's Jacksonian Prize Essay for 1923. It is based on "a review of the literature and a study of forty hitherto unreported cases of this disease". After a brief account of the anatomy and development of the testicle, the author gives an excellent description of the morbid anatomy and histology of testicular new growths. It is pleasant to find that his views tend towards simplification rather than elaboration, for he regards the malignant new growths as virtually of two kinds only, namely, malignant teratoma and spheroidal-celled carcinoma, these occurring with approximately the same frequency. Sarcoma also occurs, but it is so rare as to be almost

negligible. The confusion and uncertainty which have hitherto existed concerning these tumours the author attributes to the many forms of new growth which may arise from the different elements of a teratoma. In considering the question as to the predisposition of a misplaced testicle to malignant disease, the conclusion is reached that this predisposition probably does exist, but has been exaggerated. In his discussion of the differential diagnosis of tumours of the testicle the author insists on the extreme difficulty which may occur in distinguishing a malignant new growth from a gumma. The distinction may be impossible to make, and early operative investigation is urged when the doubt arises.

The results of simple orchidectomy for malignant new growth are stated to be very bad, a certain mortality of over 80 per cent being found. The author therefore advocates the more frequent performance of a radical operation, entailing the removal of the lymphatic channels from the testicle, together with the pre-aortic glands, by a retroperitoneal route. His own experience of this operation at present includes only three cases.

The volume is excellently illustrated with microphotographs and coloured drawings by Mr. H. Mockridge. It should help to shed light in a somewhat dark corner of surgical pathology.

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*Die Chirurgie.* By a number of contributors. Edited by Professor M. KIRSCHNER (Königsberg) and Professor O. NORDMANN (Berlin). Six volumes. Each volume to be published in several parts. To be completed in about two years. Imperial 8vo. With numerous figures in the text and many coloured plates. Berlin and Vienna: Urban and Schwarzenberg. Each part, sewed M.10, bound M.25.

THIS monumental work, of which four parts (forming portions of Vols. I, III, and VI) have already appeared, promises to be a complete account of general and special surgery, written by upwards of sixty German surgeons with the industry and completeness so characteristic of their race. It will form an invaluable storehouse of information and be a standard text-book of reference.

Part I, pp. 396 with 193 figures in the text and 2 coloured plates, opens with a general historical introduction by Brunn, taking each section of surgery in turn. It is noticeable that the account of modern surgical development makes little or no reference to any but German workers. Leusden deals with anæsthesia, local and general, laying very great stress on the former.

Boit writes the section on aseptic and antiseptic surgery, including an account of the arrangement of the operating room. Kurtzahn describes the application of the Röntgen rays and radium for purposes of diagnosis and treatment. There are many good figures illustrating typical injuries and diseases, with coloured plates of X-ray lesions. The technique and scope of radium therapy is well described. Bauer deals with constitutional conditions. He gives an elaborate discussion of the problems of heredity, and this is followed by an account of the rôle of the endocrine glands and of diabetes in relation to surgery.

The second Part published, pp. 386, with 215 figures and 14 coloured plates, will form a section of Vol. III. It deals with the lymph-glands, the breast, the glands of internal secretion, and the pituitary. Neupert takes the first of these subjects, and gives a thorough account of the lymph-gland system from the structural and pathological points of view. The anatomical distribution of the glands is very briefly described, and presumably will be included in various special subjects. The coloured figure in this article is unaccompanied by any legend, and it is not easy to find reference to it in the text. The surgery of the breast is by Klose and Sebening, and constitutes a most valuable and complete monograph, with 13 coloured plates in addition to 122 figures in the text. The anatomy and pathology are particularly well described, and the illustrations are complete and artistic. The debatable question of the spread of cancer is hardly touched upon. It is said to spread chiefly by the lymphatics and to elect the vertebral bodies and neck of the femur as favourite sites for metastasis, but the facts of perilymphatic fibrosis, the distribution of the cutaneous nodules, and the theory of permeation are not mentioned. It surprises the English reader to find a complete account of Warren's operation

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**La Pratique chirurgicale illustrée.** By VICTOR PAUCHET. Imperial 8vo. Vol. VII, pp. 256, with 188 illustrations; Vol. VIII, pp. 250, with 265 illustrations. 1925. Paris: Gaston Doin. 30 francs net, each volume.

THESE further sections of Pauchet's work are very similar to former volumes. They contain a miscellaneous collection of operations performed by the author, and are illustrated by somewhat diagrammatic drawings of the appearances seen by the artist during the actual course of the operations. In Vol. VII the first two chapters are written by other surgeons: the treatment of facial wrinkles by Virenque, and the technique of staphylorrhaphy by Victor Veau. The obliteration of the wrinkles of age by surgery will not appeal to Englishmen, but the chapter on cleft palate is interesting, as some ingenious instruments and suturing methods are advocated. It is recommended to suture the palate before the end of the first year unless the cleft is very wide. Pauchet writes a clear description of an extensive operation for a malignant thyroid, after which the non-operative treatment of fractures of the upper extremity is dealt with. Some of the methods differ from ours, but they always appear logical, though the apparatus seems rather cumbersome on occasion. The author recommends leaving the head of the humerus in its dislocated position when a fracture of the anatomical neck occurs in association with a dislocation of the shoulder.

It is not surprising to find Pauchet returning again to the subject of gastric surgery both here and in Vol. VIII. Much of the ulcer surgery is dealt with in former volumes, but cancer of the stomach is more fully described than elsewhere. Fifty per cent of the cases coming to him are suitable for surgery. The mortality after operations for cancer of the stomach is 20 per cent, but 10 per cent live more than ten years. At the end of Vol. VII is rather an original way of treating a perforated duodenal ulcer. In Vol. VIII methods of skin-grafting are described. There is a good chapter on goitre surgery, with remarks on the use of X rays in treatment. Salivary fistulæ, umbilical hernia, undescended testicle, and excision of the colon form short sections. The operation for intestinal obstruction seems to require too much exposure of the intestine, and too much manipulation, to be entirely satisfactory. Excision of cancer of the prostate forms a very interesting chapter and is well worth perusal.

This atlas of surgical operations is unique. The illustrations are wonderfully clear. The author's activities are many-sided, and surgery in his hands is evidently pushed to its limits. The books form an extraordinary record of one man's work.

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**Traitement des Fractures et Luxations des Membres.** By JACQUES LEVEUF, Chir. des Hôpitaux de Paris, and CH. GIRODE, P. MORNARD, and RAOUL MONOD, Chefs de Clinique à la Faculté de Médecine de Paris. With a Preface by Professor PIERRE DELBET. Crown 8vo. Pp. 464, with 247 illustrations. Paris: Masson et Cie. Fr. 25.

THIS is an interesting and valuable book, because, emanating from a group of younger Paris surgeons, and inspired by the idea of Delbet's master mind, it may be taken as a fair representation of the French hospital practice.

It is divided into two parts. The first deals with non-operative methods, manipulation, splinting, and after-care. The details of the apparatus used differ from those we are accustomed to, though the main principles are the same. Delbet's traction apparatus for fractured femur is given great prominence. To us it seems difficult to believe that this apparatus can give any precision, and it must require much experience before it can be used safely.

The second portion of the book is devoted to the open operations for fractures. In it the teaching and practice of Lambotte is followed closely. The various methods of Parham's bands alone and with plates are also given some prominence. Apparently the French surgeons are entirely satisfied with the metallic fixation of broken bones. They appear to be almost unaware of the use of bone nails, screws, pegs, or grafts. For example, in fracture of the neck of the femur a long carpenter's screw is depicted, whereas the bone nail, which gives a firmer fixation and becomes incorporated in the tissues, is ignored. The book, in regard both to text and illustrations, is a model of brevity and clearness.



**Praktikum der Chirurgie.** By Professor Dr. O. NORDMANN. Third edition. Royal 8vo. Pp. 796, with many illustrations. 1925. Berlin: Urban and Schwarzenberg. Sewed, M. 33; bound, M. 37-50.

This is a large book of a kind a little different from any we have in this country. The author does not give systematic accounts of the different diseases, but confines himself to accounts of the clinical features and diagnostic difficulties met with at the bedside. Treatment which is ordinarily carried out by residents, and emergency operations, are fully described, but other major operating is indicated only. It is interesting to note that defibrinated blood is used as often as citrated blood for transfusion. We observe that Murphy's drip method of proctoclysis is referred to as Katzenstein's, and the procedure described ignores Murphy's insistence on the fact that the conducting tube should be unobstructed by any clamp. In the section on appendicitis the author rightly, we think, recommends that, in general, drainage only of a localized abscess should be done. With regard to the habit of closure of the abdomen when diffuse peritonitis is present, he adopts an attitude of caution, believing that it should not be done as a routine, but only in carefully-selected cases. Cholecystectomy is performed from the fundus towards the neck, a method which has a good deal in its favour. After any laparotomy, if signs of diffuse peritoneal infection occur, Nordmann says that any treatment may be carried out except re-laparotomy, which is invariably followed by death. Altogether the book seems a reliable guide for the student and emergency surgeon.

**Diagnostik mit Freiem Auge.** By Dr. EDUARD WEISZ. Foreword by Professor Dr. FRIEDRICH KRAUS. Second edition. Royal 8vo. Pp. 178, illustrated. 1925. Berlin: Urban and Schwarzenberg. Unbound, M. 7-20; bound, M. 8-70.

This is a monograph of 178 pages which might easily have been compressed into a smaller compass. It deals solely with a method of diagnosis called ectoscopy, which consists in the careful observation of the respiratory movements that can be seen in the intercostal spaces, neck, and abdomen. Several ways of carrying it out are described: inspection in quiet breathing, inspection on quicker but not deep breathing, the speech and sniffing phenomena. There is no doubt that the author has done a service by calling attention to the necessity for accurate observation, which indeed all the older clinicians insisted on; but whether this method is or will be of great use may be doubted. He affirms that the eye becomes skilful with training, and can observe slight movements which at first were undetectable, that the lower limits of the lungs can be seen, and that help is gained in many thoracic conditions and in some abdominal discases. It is careful and interesting work, the worth of which only a trial can demonstrate.

**Methods in Surgery.** By GLOVER H. COPPER, M.D., Instructor in Surgery, Washington University School of Medicine; Clinical Assistant to Barnes Hospital; Surgeon to the Washington University Dispensary and St. Louis City Hospital. Crown 8vo. Pp. 232. 1925. London: Henry Kimpton. 14s. net.

This book is a guide for house surgeons, more particularly those working in three Washington hospitals. Its interest lies in the insight it gives into the organization of these hospitals, especially the arrangements for the keeping or consultation of clinical notes, for obtaining radiographic, chemical, and other examinations, and the instructions given to the residents. Some of these latter appear strange to us, as, for example, the admonition not to wear their hats in the laboratories and not to ask fees from the patients. The different diets classified according to their caloric value are useful. The directions given for pre- and post-operative treatment are rather lacking in detail. There is nothing very striking about the book.

**Guy's Hospital Reports.** Vol. LXXV, No. 3. July 1925. Edited by ARTHUR F. HURST, M.D. Royal 8vo. Pp. 124. London: Wakley & Son (1912) Ltd. Annual subscription, £2 2s. net; single members, 12s. 6d. net.

The reports of Guy's Hospital continue to maintain their interest, which is remarkable considering the very large number of medical journals that are published throughout

the year. To those interested in *Kiats* there is a delightful article which will appeal, entitled "*Kiats as a Medical Student*", written by Sir William Hale-White. There are two articles on oesophageal diverticula which are of surgical interest; they refer to those rare cases of pouching of the lower end of the oesophagus.

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*Chirurgische Operationslehre*. By Professor FRIEDRICH PELS LEUSDEN. Fourth edition. Royal 8vo. Pp. 832 with 771 illustrations. Berlin and Vienna: Urban and Schwarzenberg. Sewed. Dlist. bound. 1121.

This is a text-book on operative surgery similar to those published in most languages. As it is a paper-covered book written in German, and does not contain any specially new matter, it does not appear likely to command itself to the English reader.

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*Scritti medici in Onore di Antonio Carle*. In two volumes. Royal 8vo. Pp. 756 and 765. Illustrated. 1925. Bologna: Lidoio Cappelli. Each, Lire 20.

VOLUMES XII and XIII of the *Italian Archives of Surgery* are devoted to contributions from a great number—more than a hundred—of Italian surgeons, as a tribute to the work and teaching of Antonio Carle. Many of the most famous names in contemporary Italian surgery are included, and the 1560 pages together give a brilliant picture of their present-day activities. Not all the articles are of great importance: some are little more than careful records of remarkable cases; some of the more important are not entirely new—for example, the beautifully illustrated report of his experimental work on acute pancreatitis by Calzavara; but as a whole it is a splendid witness, by pupils and friends, of the esteem in which Carle's thirty years of teaching is held.

It is impossible to review, even cursorily, more than a fraction of the contents. Professor Donati contributes to the study of traumatic shock an experimental investigation into the method of action of toxic substances derived from contused tissues. Lattes throws doubt on the limitation of blood-groups to four. There are several studies of grafts of formalin-fixed tissues. Professor Purpura writes on blastomycosis. Of two or three papers on echinococcus, one, Nasseti's, suggests a method of treating the cyst wall similar to Matas' treatment of aneurysm. Palmieri describes a method of indirect X-ray irradiation which permits 'cross-fire' attack from a single source. There are reports of investigations into the presence of organisms in the bone medulla of simple fractures, and in the epiphyses (Fiori and Uffreduzzi). Cranial and cerebral surgery are represented by, among others, Bufalini and Solaro. Bertolotti prints some beautiful radiograms of cranio-pharyngeal tumours. Torraca records a case of total necrosis of the mandible, and de Gaetano discusses the operative treatment of ankylosis of the jaw.

In the second volume Parlavecchio indicates the technique of partial and complete resection of the sternum; Anzilotti deals with the cure of bronchial fistulae, insisting that complete liberation of the lung from the chest wall is the *sine qua non* of success. Vespignani, who writes on the direct imprint of the left costal arcade on the stomach, and its importance in reading opaque-meal appearances, is not very convincing. Costatini figures a plastic method of overcoming the difficulties encountered in excising gastric ulcers near the cardia. Gastric function and peptic ulcers are the subjects of half a dozen papers; the biliary tract and function of another group; in the renal field, amongst others, Stropeni describes experiments on the influence of decapsulation on the renal circulation. Muscatello relates a case, said to be the first, of inveterate horizontal luxation of the humerus. Durante records a case of periarterial sympathectomy of the posterior tibial after failure of a similar operation on the femoral; Scalone (in Vol. I) describes his technique for this operation; his diagrams are, to say the least, optimistic. There are several useful orthopaedic contributions.

The standard of the work is high, and the field covered vast. Professor Carle must indeed feel himself honoured.

*Peritoniti acute.* By DOTT. V. PUCCINELLI. Preface by Professor RAFFAELE BASTIANELLI. Imperial 8vo. Pp. 281, illustrated. 1924. Bologna: Licinio Cappelli. Lire 35.

This book is honoured by a preface from the pen of Professor Bastianelli, a full record of whose cases from 1900 to 1923 appears in the second part.

The first part, 92 pages, is devoted to a review of recently expressed views from all sources on the physiology, pathogeny, histology (well illustrated by photomicrographs and drawings from the author's specimens), pathology, symptomatology, prognosis, and treatment of peritonitis in its various forms. There is also a short section describing new experiments on absorption from the peritoneal cavity.

The second, statistical, part is of course valuable, for it contains short notes of 1005 operation cases, of which 484 were due to war wounds. Peritonitis due to perforation of the appendix shows a mortality of 27.4 per cent; but it must be remembered that cases as far back as 1900 are included, and that patients still arrive at hospital much too late. Professor Bastianelli deplores the ill success of all his efforts to educate the public and the profession in the supreme importance of promptitude in resorting to operation. Perforated peptic ulcer, mortality 37 per cent; typhoid perforation, 73 per cent; perforation of tuberculous ulcer, 63 per cent; 'diplococcal' peritonitis, 5 out of 7 cases; puerperal peritonitis, 54 per cent; gynæcological, i.e., chiefly salpingitis, 29 per cent: this gives a total mortality of 42 per cent in 412 consecutive cases of peritonitis other than that due to wounds.

The third section of the book, more than sixty pages, is devoted to bibliography.

*Operative Surgery.* By J. SHELTON HORSLEY, M.D., F.A.C.S., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va. Second edition. Imperial 8vo. Pp. 771, with 666 illustrations. 1924. London: Henry Kimpton. 52s. 6d. net.

THE first edition of this work appeared in 1921, so that the need for a second within three years speaks well for its popularity. As pointed out in the preface of the first edition, the author does not intend that the book should cover the whole field of operative surgery. The operations which are described are those which he is in the habit of performing, or are those which he considers to be best suited to the condition needing surgical interference.

A special feature of the present edition is the inclusion of interesting new operations which, so far as the author is aware, have not hitherto appeared in either text-book or monograph. These operations are: the lymphaticostomy of Costain, Storkey's operation for innervating paralysed muscles, Finney's pylorotomy, Graham's pulmonary lobectomy, Cutler's valvotomy for mitral stenosis, the operation of Coffey and Brown for angina pectoris, Frazier's chordotomy, and Kerr's method of intestinal resection. All of these operations are carefully described, and form a valuable addition to the book.

The work has been brought thoroughly up to date, and maintains the high grade of excellence displayed in the first edition. We can cordially recommend a perusal of its pages to all those who are desirous of acquainting themselves with the best methods of American operative surgery.

*Chirurgie de l'Appareil urinaire et de l'Appareil génital de l'Homme.* By PIERRE DEVAL and J. GATELLIER. Sixth edition. Crown 8vo. Pp. 284, with 310 illustrations. 1924. Paris: Masson & Cie. Sewed 12 fr., bound 15 fr.

THIS book is one of the series of *Précis de Technique opératoire*; it is the work of the Prosectors of the Faculty of Medicine of Paris, and represents their teaching of operative surgery. It contains 281 pages and is a miracle of conciseness; in addition to the 310 illustrations, there is an excellent description of the operations commonly performed on the kidney, the ureter, the bladder and prostate, the urethra, and the testicle—and the book can be comfortably carried in one's pocket. The numerous illustrations are in black and white, and not only show the various operations, but in some cases depict the anatomy of the parts concerned; they are extremely well done, and are a real help to the understanding of the text.

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As the work is intended to be a handbook for a course of operative surgery, there is no clinical information, but as a manual of surgical technique for a special department of surgery it is very good, and we can recommend it to our readers.

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**La Secrezione gastrica.** By Dott. ANTONIO CIMINATA. Royal 8vo. Pp. 247, illustrated. 1925. Bologna: Licinio Cappelli. Lire 35.

THE admirable series of monographs published by the firm of Licinio Cappelli at Bologna deserves special mention for its excellent typography, the high standard of its illustrations, and its freedom from errata, as well as for the character of the work itself. This volume is a good example of the work being done in the Italian universities. In the main it is a record of a large series of experiments on dogs, in continuation and confirmation of the work of Heidenhain and of Pawlow, which is described and discussed, together with other work on the same subject, in the first section of the book. The author gives a full account of his method of making a small attached but separate stomach for investigation of the secretions, and of the very numerous experiments conducted.

Among the results he observes are the following: a direct relation between the percentage of water in the food and the quantity of juice; a direct relation between quantity of succus and period of latency; the period of maximum secretion and the duration of secretion vary with the type of food; water, by itself, stimulates a flow, but it is scanty and of brief duration; the degree of acidity corresponds to velocity of secretion, not to type of food; peptic value corresponds to the type of ingesta.

He concludes that the process of secretion considered as a whole is an adaptation of glandular activity to the qualities of the food; that most foods contain in some degree chemical stimulants of secretion, which are developed in the earliest stage of digestion; the first flow of juice necessary to start their production is occasioned by psychic reflex—appetite, in fact—and perhaps by water itself. The psychic reflex reaches the stomach by the vagi; the subsequent chemical stimulus, equally 'nervous', acts, however, directly on the plexuses in the stomach walls. Fats inhibit chemical stimuli. Acidity does not vary directly with different foods; the degree is determined by the amount of neutralizing mucus, and that depends on the rate of flow of succus; 'hyperacidity' merely means a not-yet-neutralized succus, a failure of equilibrium. It is extremely doubtful if succus secretion is in any way influenced by substances reaching the cells by the blood. There is a bibliography.

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**Megacolon congenito.** By Professor GUALFARDO TONNINI. Imperial 8vo. Pp. 243, illustrated. 1924. Bologna: Licinio Cappelli. Lire 20.

THIS monograph covers the whole history and literature of Hirschsprung's disease and its allied conditions. It gives a résumé of 63 cases from Italian publications besides details of a personal case. It suffers, like so many similar Italian books, from a plethora of diverse descriptions and opinions, amid which it is difficult to discover what are the author's own conclusions; there is evident reluctance to assess the value of the diverse views. Probably the truth is that congenital megacolon is not one condition but many; and if this book contributes, as it should, to their disentanglement and discrimination, it may be possible to standardize treatment. Very little is said here, except in the author's own case report, about radiological investigation, yet the condition is pre-eminently one for such study; the passage of an opaque meal and the flow of an enema ought to reveal a good deal as to the site and character of the 'obstruction', and give valuable information for determining the type of operative treatment best adapted to cure. There is an extensive bibliography.

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## EPONYMS.

By SIR D'ARCY POWER, K.B.E., LONDON.

### XX. THOMAS'S HIP SPLINT (*continued*).

THOMAS's hip splint seems to have been in use for about twenty years before Professor Rushton Parker, of Liverpool, seeing it by accident and at once appreciating its value, insisted that the inventor should publish an account of it. Thomas, as has been shown, had no vanity, no secretiveness, and above all no desire for publicity. He was wrapped up in his work, he always had more to do than was good for his delicate frame, and, as his involved style shows, he had but little skill with his pen. His disinclination to make his methods more widely known was due partly to want of time and partly to a modest belief that he had done nothing unusual by the invention of a splint which has revolutionized the treatment of fractures in bones and diseases in joints.

The first published account of the splint appears in *Diseases of the Hip, Knee, and Ankle Joints and their Treatment by a New and Efficient Method*, by Hugh Owen Thomas, Liverpool; published by T. Dobbs & Co., 69 Gill Street; 8vo, pp. iv and 101, illustrated with 17 plates. Messrs. Dobbs & Co. were a small firm of printers, friends of Thomas, and the book was practically issued privately at the price of ten shillings. It was dedicated to "my friend and respected fellow Townsman Dr. F. Ayrton as an acknowledgment though an inadequate one of many valuable services rendered to the Author." Dr. Ayrton had been resident medical officer at the Liverpool Fever Hospital and afterwards Lecturer on Medical Jurisprudence at the Liverpool School of Medicine. The Preface to the book is dated from 11 Nelson Street, Great George Square, Liverpool, July, 1875. A second and enlarged edition was published in 1876. I quote from the rare first edition, a copy of which is in the Library of the Royal Society of Medicine. Thomas writes at p. 18:—

"There is an opinion prevalent, that only gentlemen on the staff of our public charities can treat, with any chance of success, this affection [hip-joint inflammation] and certainly, hitherto, they have had advantages not possessed by the general practitioner, having at command the wealth of the

charity, to which they are attached, and being thereby enabled to order the costly appliances at present in use.

"Persons living at a distance from large towns, rarely receive professional assistance, as the one thing supposed to be needful cannot be obtained at home. This has induced me to describe such details, as will enable the surgeon to treat his cases at home, with no more mechanical assistance than can be rendered by the village blacksmith and saddler, and the poorer class of sufferers will, at a small cost, be assisted as effectually as the wealthier classes.

"We will suppose the patient, a boy of about 10 years of age, having been examined, and the affection diagnosed as disease of the right hip joint, the surgeon (*then*)\* proceeds to measure him for the instrument (*suitable for a case in the early stage*). He requests the patient to stand on the left limb (the one supposed to be sound), and then places under the sole of the right foot a block or a book, one inch thick, telling him to rest the foot of the affected limb on it. If the spine is then straight he is ready to have the contour of the sound limb (*and portion of the trunk*) taken. If the spine is not of normal line, then add another block, or several blocks, (*if considered necessary*) until the unsound limb is raised sufficiently to allow the spine to resume its natural condition. Now take a long flat piece of malleable iron, one inch by a quarter for an adult, and three quarters of an inch by a quarter (*three sixteenths*) for children, and long enough to extend from the lower angle of the shoulder blade, in a perpendicular line downwards over the lumbar region, across the pelvis slightly external (*but close*) to the posterior superior spinous process of ilium, and the prominence of the buttock, along the course of the sciatic nerve to a point slightly internal to the centre of the extremity of the calf of the leg. The iron must be carefully modelled to this track, to avoid excoriations which would otherwise trouble the patient during treatment. (*In neglected and extreme deformities, under such conditions, the model is taken from the deformed limb, and, as the muscles relax the appliance is altered by aid of the wrenches. The lumbar portion of the upright must be invariably almost a plane surface, rotated on its axis more or less in proportion to the plumpness of the patient.*) This iron forms the upright portion. It is also very necessary that this upright should come below the knee, to enable the surgeon to fix this joint, otherwise the patient would flex the knee, and rising the leg as a lever, would strain the hip-joint. Then measure round the chest, a little below the axilla, deducting, in the case of an adult, three (*four*) inches from the chest circumference. This latter will be the measure for the upper cross piece, which is made from a piece of hoop iron, one and a half inch by one eighth of an inch. The hoop iron is firmly jointed with a rivet to the top of the upright. At one third of its length from the end next to the diseased side is the upper ring modelled to the outline of the trunk, which is oval in shape. It is important to give the upper crescent this oval shape, otherwise the machine will rotate from its position behind the body, also inversion of the limb will occur. Another strap of hoop metal five sixteenths (*three quarters*) of an inch by one eighth of an inch, and in length half (*two thirds*)

\* The words here printed in italics appear in the second but not in the first edition.

the circumference of the thigh, is fastened to the upright, at a position from one to two inches below the fold of the buttock, according to the age of the patient, then another piece of metal of like strength, equal to half the circumference of the leg at the calf, is firmly rivetted to the lower extremity of the upright. The points of junction of the cross pieces with the upright should be out of centre. The short portion of the top half circle is next to the diseased side (*with a space intervening*), while the long portion must be closely fitted to the sound side. In my earlier experience the upper crescent (*a second crescent*) embraced the pelvis instead of the chest, but I found it very inefficient, difficult of application and painful to wear. (*If the machine should tend to rotate from the diseased side, then daily contract the long wing of the crescents, and expand the short ones; or should it tend towards the spine TOO FAR, then reverse this manipulation until the appliance becomes set in the correct position.* In applying an instrument with two uprights, care should be taken to measure the distance between the tip of right and left posterior spinous processes, and then to set the uprights parallel and apart, one inch more than such measurement, or it cannot be tolerated by the patient. The two uprights should be connected by a crossbar, when practicable, which is not possible when the double instrument is used for reduction of deformities; this crossbar when used, will be found useful for the attendant to grasp in nursing. This appliance, with two uprights, is indispensable when both joints are affected, and if the patient or his friends do not object, will be found easier of application, and therefore of more certain efficacy, even in cases where only one articulation is affected.)



FIG. 404.—Thomas hip splint.

Reproduced from *Diseases of the Hip, Knee and Ankle-joints*, by Hugh Owen Thomas, Liverpool, 1875.

"The instrument is now ready to be padded and covered by the saddler with basil leather. (*The instrument is now ready to be padded and covered. The former is conveniently done with boiler felt (No. 1 thickness,) which should*

*not be used in more than a single layer ; the latter is done by a saddler with basil leather. I recommend this quality of leather as it never becomes offensive with the patient's secretions, &c., which is the case with chamois leather so frequently used in the construction of surgical appliances.)*

"However correctly it may have been modelled, it will often occur that some slight alteration will be demanded, when it comes to be applied to the patient, either on the first day, or at some period during the progress of the case, or the case may be one of long duration, uncontrolled, and consequently be attended with much deformity, then the surgeon may have weekly to alter the curves &c. of the appliance. To enable him to do this, I have devised and used a set of instruments and wrenches. By the aid of these the surgeon is always independent of any mechanical (cutler, &c.) aid until the case has terminated, or he may remodel an old appliance to use again. The patient being placed in the machine, a strap and buckle close the upper circle round the chest, and the limb is bound with flannel from the calf upwards, beyond the small crescent as is shown in *Fig. [404].*"

"When applied correctly the long portion of the upper crescent should be close to the trunk (*and should exercise some pressure on the sound side,*) the short portion a little space from the trunk. This is necessary to hinder rotation of the instrument, and the upright stem should have a perceptible rotation outwards, and be fitted so that it passes to the inner side of the popliteal space ; this will avoid rotation inwards of the limb, a defect easily avoided by attending to these details.

*"(Should the instrument rotate towards the diseased side, and so become a side splint, the surgeon should contract the longest wing of the upper crescent and expand the shorter one ; or if the instrument does not rotate, yet the stem is not over the prominence of the buttock and well behind the thigh, then the upright requires more twisting.)*

"It is advisable that the sufferer should be confined to bed for a short period, at the commencement of the treatment, until the night pains and disturbed sleep have ceased. This is the first stage of the mechanical treatment. (*It is preferable to place the patient in a soft bed, during the first stage, rather than on a mattress, which is objectionable granting that the surgeon has not selected the iron of too slight proportion, an error I notice many are inclined to commit.*

*"The hip appliance if not moulded by the surgeon so as to remain continually behind the trunk, and painless to wear, then it requires more of the surgeon's skill and perseverance, or it would be useless to the patient. A few minutes of interruption is as great an evil as so many days, and would not be uninterrupted rest, which is so essential to success.*

*"It is very advisable that the sufferer should be confined to bed for a period, at the commencement of the treatment. This preliminary reclusion, I have never noticed to injure the general health, but invariably improves the patient's condition, and shortens the acute stage. During the first stage of the mechanical treatment, the surgeon being satisfied that suppuration has been avoided, he permits the patient to proceed on to the second stage.)*

"Next comes the second stage. The patient is allowed to go about with the assistance of crutches, the frame continued, and an iron patten (*at least*



*four inches in depth*) placed under the shoe of the sound limb. This must be continued until the limb is well atrophied, around the great trochanter (*the outline of which should be more discernable than that of the sound side.*)

"We now come to the third stage. The patient takes off the framework in bed, and replaces it during the day, still using the crutch and patten for a certain period.

"We now arrive at the fourth stage. The patient discards totally the frame, and uses the crutch and patten only. These he sets aside after the surgeon is well satisfied of the permanence of the cure. If the case does not progress to the surgeon's satisfaction, some of these stages must be prolonged.

"The weight of the limb is equal to reducing any angular deformity of the lower extremity (*not resulting from true ankylosis*), and capable also, in a slight degree, of diminishing any shortening, should absorption of the head of the bone occur,—provided a suitable mechanical arrangement is applied (*and continued during a sufficient length of time*).

"The splint ought to be applied at once, whatever the stage of the disease. Forcible flexion, extension, tenotomy, or chloroform &c., are to be avoided as injurious (*unnecessary*). In the presence of this method, these operations are objectionable (*undesirable*), though they were essential at one time. Even should the deformity be an extreme one, no violence must be attempted; the limb must be gently persuaded to come back from the erring position, and as the limb assents, the wrenches should be used to alter the hip instrument towards the normal lines."

The persistence in treatment until there is wasting of the glutæus, and the *assenting* of the diseased limb, are curiously reminiscent of John Hunter's method of keen observation and thinking.

## CHORIONIC CARCINOMA IN THE TESTICLE: WITH A REPORT OF A NEW CASE.

BY R. M. HANDFIELD-JONES, LONDON.

### HISTORICAL SUMMARY.

THE first recorded case of chorionic carcinoma in the uterus came from Sanger in 1889, followed independently by Pfeiffer in 1890. In view of the latter article, Chiari revised some of his previous cases and brought them into line with this new tumour. All these observers considered their specimens to be decidual sarcomata; but in 1895 Marchand first recognized their epithelial origin, and although he at first regarded the syncytium as maternal, he recognized later that it was of foetal origin also.

To French pathologists belongs the honour of recognizing similar structures in the testicle, for in 1878 Malassez and Monod<sup>34</sup> reported a case, followed in 1898 by Carnot and Marie,<sup>8</sup> and in 1900 by Dopter.<sup>14</sup> In these cases the tissue attracting particular attention was the multinucleate giant-celled masses. They noticed the intimate relationship of these masses to the blood-vessels, and described the vacuolation with blood-corpuscles in the spaces. Though other cells were seen and described, little attention seems to have been paid them, and the tumour was considered to be of vaso-formative nature, and named the 'sarcome angioplastique'. Early in 1900 Wlassow<sup>75</sup> added four cases, and, reading his description in the light of modern knowledge, their classification as chorionic carcinoma presents no difficulty. In one of his cases the splenic metastases showed syncytium with ciliated borders, and in two of his cases typical areas of epiblastic and mesoblastic derivatives (cartilage, muscle, and squamous epithelium) were found, but because he could recognize no hypoblastic structure he refused to accept their teratomatous origin. Although he realized the close similarity between his cases and the chorionic carcinoma of the female, yet he failed to proceed to the logical conclusion, instead of which he named them 'epithelioma syncyomatodes testiculi'. It was with the ground thus well prepared for him, that Schlagenhauser,<sup>59</sup> in 1902, finally placed these cases on a firm foundation. He pointed out the many features of these growths which exactly coincided with those seen in the uterine tumours, and going further maintained that they were all derivatives of a teratoma. He believed that such a teratoma contained rudimentary foetal membranes which were responsible for the development of the chorionic carcinoma. This argument seems unfounded, and in later years Pick<sup>48-53</sup> has demonstrated its origin from a neuro-epithelial tube, and R. T. Frank<sup>19</sup> from an epithelial-lined cyst. Nevertheless, Schlagenhauser's main contentions remain unquestioned to-day, and that the resemblance of these tumours to the female type is more than a superficial one is proved by the fact that in three recorded cases activity of the breast with colostrum secretion was a noticeable feature (Cooke,<sup>12</sup> Warthin<sup>73</sup> and Gabarini<sup>77</sup>).

FIG. 405.—Chorionic carcinoma of the testicle. One half of the testicle of the case reported. The body of the testicle is replaced completely by growth. The epididymis is normal. The cord is not infiltrated, but is enlarged from vascular hypertrophy. In the lower part of the growth is an area of blood clot, and it is around this that the best histological preparations of chorionic carcinoma have been obtained. ( $\times \frac{3}{4}$ .)



FIG. 406.—Secondary growths in the lung. This specimen is No. 754A in the Westminster Hospital Museum. The appearances are so exactly similar to the lungs of the author's own case that the latter were not drawn. Many of the nodules are seen to be typical plum-coloured areas, but in addition there are pinkish growths not infrequently seen in multiple metastases. ( $\times \frac{3}{4}$ .)

## THE NAKED-EYE APPEARANCE.

The appearance will depend upon the amount of ordinary teratomatous structure present. If in any recognizable quantity it will present the typical picture with small cysts, areas of blue translucent cartilage, and so on. The area of chorionic carcinoma looks more like blood-clot than anything else. Plum-coloured areas are seen, practically homogeneous throughout, save for small, white, friable areas of necrosis. In my specimen (*Fig. 405*) the general appearance is that of a teratoma, but there is one area of blood-clot around which the best microscopic parts of the chorionic growth are to be found. In the fine specimen in the Westminster Hospital Museum the whole testicle is replaced by the reddish, plum-coloured growth, with a few strands of fibrous tissue. The metastases usually present the same red areas, but occasionally (*Fig. 406*) they are not stained with blood, and are then white homogeneous nodules.

## THE MICROSCOPIC FINDINGS.

It is necessary to insist that the diagnosis of chorionic carcinoma depends upon the recognition of certain definite histological characteristics. There are several cases in the literature described as chorionic growths on quite inadequate grounds; there are preparations which arouse suspicion, but in which conclusive evidence is lacking. Again, of recent years certain observers, notably in France, have suggested that the diagnosis can be made in the presence of only one of the recognized elements of these chorionic growths. In the event, these cases may be regarded with suspicion, but no proof is present, and a confident diagnosis has no foundation. It seems desirable that the appearances in the testicle should be restated, for in no other organ does such unfortunate confusion exist as to the classification of malignant growths, and the issue can only be further confused if unwarrantable subdivisions of the chorionic carcinoma are allowed to creep in.

Marchand<sup>35-38</sup> has described two forms, the typical and the atypical, and he can hardly be improved upon. The following is the translation from R. T. Frank's<sup>19</sup> most admirable paper:—

“In the *typical* group the characteristics of the chorionic epithelium, which appears in the first stage of gestation, are represented with few or no variations. They show well-developed continuous syncytial masses of irregular multinucleated strands and branching protoplasmic buds, and with a more or less well-developed ground work of numerous transparent polyhedral cells of the nature of the Zellschicht (*viz.*, Langhans' cells).

“In the *atypical* group, nearly everywhere the epithelium has lost its peculiar normal grouping, and occurs only in isolated cells which may be of various forms. The cell masses which form the surface and inner layers of the decidua basalis in a hydatidiform mole may be taken as an example or pattern of these forms. These cells rarely show the habitus of the delicate transparent, membranous, and often sharply circumscribed Zellschicht element with regular oval nuclei, frequent mitoses, and glycogenic cell protoplasm. More often they are compact, more deeply staining, and very irregularly shaped cells, with nuclei varying greatly in size, sometimes attaining enormous proportions and showing intense and often equal staining properties, which allow the recognition of their syncytial character. These elements may form multinuclear aggregates, but in many cases no large syncytial masses occur.”

The elements found in chorionic carcinoma will be here briefly described. They are: (1) *The syncytium*; (2) *The Langhans' cells*; (3) *The chorionic wandering cells*; (4) *Masses of blood and fibrin*.

1. **The Syncytium.**—This consists of multinucleated protoplasmic masses of varying shape and size, and is often seen sending branching processes out into the surrounding tissues. The protoplasm stains deeply and is frequently vacuolated, red blood-cells often being seen in the vacuoles. In very well-fixed preparations the protoplasm takes on a greenish tinge with van Gieson stain, and the tint with hæmatoxylin and eosin is characteristic. The border of



FIG. 407.—Drawing from Bonney's article showing the appearances of what Marchand would classify as a 'typical' chorionic carcinoma. (Copied by kind permission from Mr. V. Bonney.)



FIG. 408.—Low-power drawing of an area seen in the reported case, in which there appears to be an attempt at the formation of chorionic villi. Flat strips of syncytium may be seen in several parts of the section, and in the centre are two masses of Langhans' cells surrounded by a very fine zone of syncytium. ( $\times 75$ .)

these masses is said to be ciliated—the stereocilia or *Bürstenaum*—but these are not true cilia. Under the high power the protoplasm often presents a foamy appearance, but this is believed to be due to commencing degeneration. The nuclei, of which there may be large numbers, are small, oval, and stain deeply and uniformly; but occasionally large spherical nuclei are seen which stain less deeply, have a definite nucleolus, and a well-marked chromatin network. Others again have no nucleolus, no chromatin network, but a series of chromatic

points. Multiplication is by direct division, and karyokinetic figures are never seen.

Syncytium has an intimate relationship to blood capillaries, and in many sections vascular channels may be seen in the process of erosion by the syncytial masses. This results in large areas of blood extravasation as more and more blood-spaces are opened up. (*Figs. 407-410.*)

**2. The Langhans' Cells.**—These are medium-sized polygonal cells with single nuclei. The cytoplasm is less dense than that of the syncytium, and generally presents a fairly clear appearance. It is very finely granular, has a wide reticulum, and does not stain deeply. The cell outline is well defined and sharp, but when present in bulk they show signs of pressure. The nuclei are larger than those of the syncytium and have a well-marked chromatin network with one or two nucleoli which stain well with nuclear stains. Multiplication is by indirect division, and karyokinetic figures are quite commonly seen. Fresh preparations sometimes when stained with Sudan III show the presence of fat, and special methods have proved that glycogen is sometimes found in these tumours. These Langhans' cells vary considerably in their arrangement, at times appearing as masses of cells mimicking a carcinoma simplex, at others lining tubules, cystic spaces, or papillary tufts.

**3. The Chorionic Wandering Cells.**—These seem to be a compromise between the two previous types. They may be mono- or multinucleated. If the former they are larger than the Langhans' cells, and if the latter they are smaller than the syncytial masses. Again, their cytoplasm stains more deeply than the Langhans' cells and less than the syncytium. The nuclei have a well-marked nuclear membrane, one or two nucleoli, and a deeply staining chromatic network. Division may be by direct or indirect methods, and according to Teacher both forms may occur in the same tumour. In the uterus these cells occur in advance of the growing edge of the main growths, and according to some authors they—rather than the syncytium—are responsible for the erosion of the blood-vessels. The exact nature and origin of these cells are quite unknown.

**4. Blood and Fibrin.**—In every chorionic carcinoma there are large areas of blood-cells in various stages of degeneration, tumour-cells dead or dying, and fibrin formation is well marked. In the outermost layers of such areas, tumour-cells may be seen with nuclei and showing fragmentation, pyknosis, and other signs of degeneracy. In some specimens this picture predominates almost throughout, and only after prolonged search can a reasonably typical area of growth be identified. This picture of masses of blood, fibrin, and necrotic tumour-cells is undoubtedly suggestive of the diagnosis, but it is not sufficient, and a typical area must first be seen.

In the testicle in a very large majority of the reported cases (*see p. 617*), teratomatous structures appeared either in the primary growth or in the metastases, and I strongly incline to the view that they do not and cannot occur in the testicle save as part of a teratoma. Again, examples are recorded which follow exactly Marchand's typical form (*see Fig. 407*, from Victor Bonney's article: nothing more typical could be needed). In some parts my tumour is absolutely typical. In other cases the arrangement is atypical, and one or other cell form may predominate. In the testicle it must be

FIG. 409.—Low-power drawing from the same tumour. The centre of the section contains a branching mass of syncytium, with many small, round, and darkly staining nuclei. Vacuoles of varying size are seen filled by red blood-cells. The granular or foamy appearance is well seen. At the upper right and lower left parts of the drawing are Langhans' cells. (Hæmatoxylin and eosin.) ( $\times 90$ .)

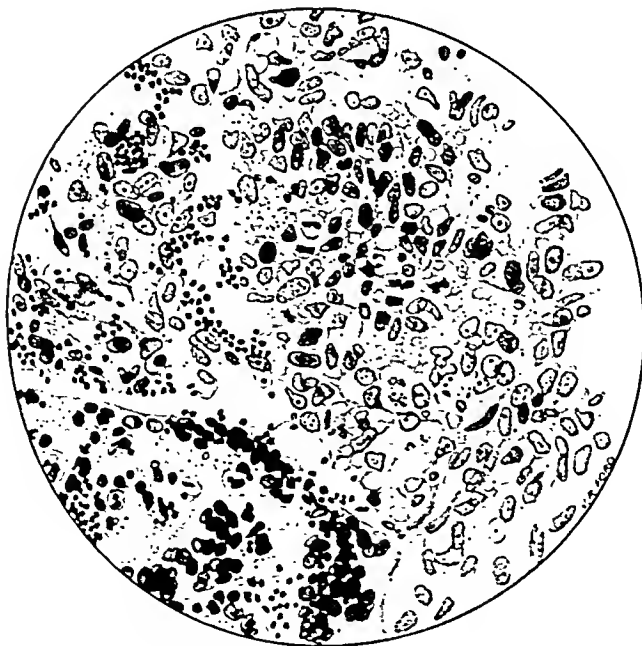
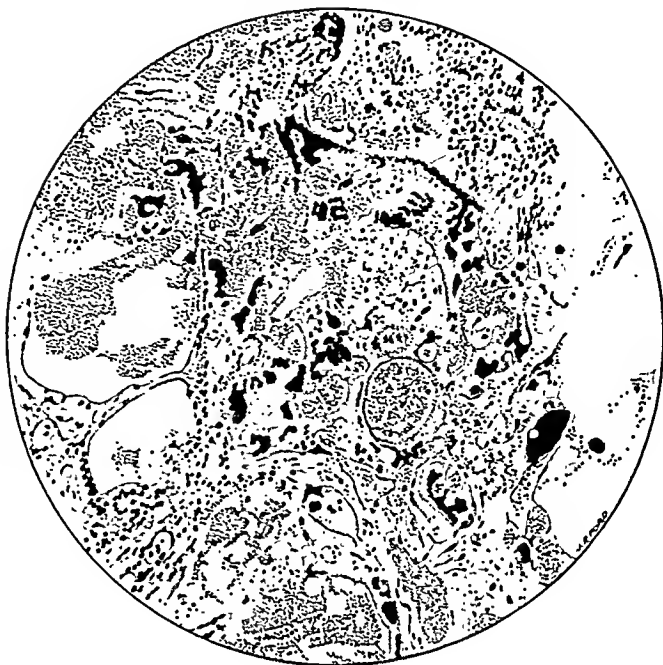


FIG. 410.—A higher magnification of the upper right-hand edge of the syncytium in Fig. 409. The character of the Langhans' cells is more clearly seen. Several mitoses are present. (Hæmatoxylin and eosin.) ( $\times 270$ )

accepted that the tendency is for these growths to be extremely atypical, and in many sections one or other element may alone be represented, and only by tracing direct continuity with a more typical area can a clear-cut diagnosis be given. And this raises a most important question. In such areas, where only one element is seen—e.g., a mass of Langhans' cells simulating a carcinoma simplex—is it possible by any histological method to say that the tissue in question is definitely of a chorionic character? Quite recently Hartmann and Peyron<sup>80</sup> have divided chorionic carcinomata in the testicle into two groups, viz., placentomata, which they describe as typical examples, and choriomata, which show no syncytium and only masses of Langhans' cells which by their arrangement mimic a carcinoma simplex. I suggest that they have brought forward no acceptable proof that these latter consist of Langhans' cells at all, and I submit once again that this is a dangerous doctrine, and liable to lead to a fresh avenue down which we may be led into worse mistakes in pathological diagnosis and a still more complicated classification of malignant disease of the testicle.

### THE METASTASES.

Chorionic carcinoma gives widespread metastases via the blood-stream, and usually this occurs at an early date. When the peculiar character of this growth is borne in mind—its intimate relationship with, and erosion of, the blood-vessels—its early dissemination is easily understood. The lungs invariably are the principal seat of secondary deposits, and later there may be a generalized distribution throughout the body. It is usual for the secondary nodules to show the same characteristics as the primary tumour, but occasionally small deposits are seen free of blood, and are then firm, white growths. They commonly show a much more typical histology than the parent growth, and in some instances they have settled a doubtful diagnosis of the primary tumour. *Fig. 408* shows the lung of a case in the Westminster Hospital Museum.

Though blood-stream dissemination occurs early and in severe degrees, this tumour also spreads by the lymphatics, and enlarged glands are commonly seen along the aorta and inferior vena cava near the renal vessels. In the case now reported, the lymphatics running into the pelvis parallel to the vas deferens were permeated by growth. This path of spread is by no means uncommon, as I have shown elsewhere.<sup>78</sup>

There is one feature of the greatest clinical interest with regard to these metastases. Certain cases are recorded of chorionic carcinoma of the uterus in which spontaneous repression of the secondaries has followed the local removal of the primary growth. As far as I am aware, no similar record exists in the testicular growths.

### THE SITE OF ORIGIN.

In the early history of this tumour many theories were advanced to explain its origin; since Schlagenhauser finally demonstrated its chorionic character, the main discussion has centred around the actual element in a teratoma that is responsible for the trophoblastic tissue. The following, briefly summarizes the various theories: (1) Malassez and Monod<sup>31</sup> regarded



it as a vasofactive endothelial sarcoma. (2) Sternberg<sup>68</sup> has recently revived the endothelial theory. (3) Monekeberg<sup>42</sup> also assigns an endothelial origin to the syncytium. (4) Conforti<sup>11</sup> suggests it is a perithelioma. (5) Schlagenhauser<sup>39</sup>—from foetal membranes in a teratoma. (6) Lubarsch—a metaplasia of embryonic ectoderm in a teratoma. (7) Pick,<sup>48</sup> Risel,<sup>57</sup> and Frank<sup>19</sup> have shown it arising from such derivatives of a teratoma as neuro-epithelium, cubical and squamous epithelium.

The generally accepted view is that it arises from some element in a teratoma, but that the formation of true foetal membranes is not necessary. Schlagenhauser's theory fails for two reasons: first, no structure in a teratoma testis has ever been described as representing foetal membranes; and, secondly, it is logical to assume that, if such membranes did exist, they should give rise to the same series of tumours that are known in the uterus, viz., the benign hydatidiform mole and the perforating hydatidiform mole, as well as the actual chorionic carcinoma. His very interesting theory would therefore become established if hydatidiform mole formation could be proved in these tumours. Five cases have been claimed as providing this proof by Breuss and Schlagenhauser,<sup>6, 39</sup> Waldeyer, Silberstein and Kanthack and Pigg.<sup>81</sup> These cases are not accepted by pathologists as a whole, and Schlagenhauser's theory as yet remains unproved.

The histological descriptions from Pick, Risel, and Frank are similar in that chorionic carcinoma has been seen in continuity with the lining membrane of certain cysts. In one case it was a neuro-epithelial tube, and in the other two cysts lined by squamous and cubical epithelium respectively. To claim that the chorionic tissue has definitely arisen from these different epithelia seems unjustifiable and contrary to the general histology of the teratomata. It is one of their characteristics that various tissues are jumbled up in a heterogeneous mass, and it is one of their commonest features to contain a cyst lined in one place by tall columnar epithelium, in another by squamous, and in yet another by low cubical, each type passing gradually or abruptly into the next. If chorionic tissue is present, it is only too likely to be found mixed up with other elements, and it is known to line cysts, part of which may be lined by another type of cell.

In the present stage of our knowledge—or rather ignorance—of the origin of the teratomata themselves, it is not possible to be assured of the finer details of their derivatives. It is enough to say that there remains little doubt that the chorionic carcinoma does not arise in the testicle apart from a teratoma, and that, until time affords a proof of Schlagenhauser's theory, we must remain content with this limited knowledge. In this paper I have described areas in the original tumour, and particularly in the cardiac metastases, which bear a striking resemblance to true chorionic villi, but I should hesitate long before considering them as proof of their origin from pre-existing foetal membranes.

#### ACTIVITY OF THE BREASTS.

Attention has recently been drawn to the activity of the male breast in connection with chorionic carcinoma of the testicle. Cases have been recorded by Cooke,<sup>12</sup> Warthin,<sup>73</sup> and Gabarini.<sup>77</sup> In each patient the breasts enlarged

and colostrum was secreted. It is a striking proof that these tumours bear a more than superficial resemblance to the chorion of pregnancy. Further, they shed an interesting light on the possibility that the teratoma may have a far-reaching influence on the general metabolism of the patient. For example, it is a somewhat intriguing thought that thyroid tissue in a teratoma may be present in such quantity as to produce a clinical hyperthyroidism.

### THE AUTHOR'S CASE.

T. W., age 40, married, a tobacconist, who had lived for several years in Australia, presented himself in the middle of April, 1925, in the out-patient department of St. Mary's Hospital, complaining of a swelling of the right testicle.

**HISTORY.**—During October, 1924, in Australia, he noticed a swelling of the right testicle, which came on quite rapidly and was accompanied by slight dragging pain in the right lower part of the abdomen. Within a few weeks he saw three practitioners, who, not believing his assertion that he had never had gonorrhœa, overhauled him with a view to establishing this diagnosis. By each he was urethroscoped, his prostate was massaged, urine examined, and numerous bacteriological examinations were made. In no instance were gonococci to be seen. The only treatment of any sort suggested was that of radiant heat to the enlarged testicle. Dissatisfied, he sailed for England and came straight away for advice. He told me after admission to the wards that he had had blood-stained sputum daily for many weeks, even while under medical examination in Australia.

**ON EXAMINATION,** he had a spherical swelling of the right testicle, perfectly homogeneous, firm but not very hard (*see Fig. 405*). The cord was definitely enlarged, but the vas was not thickened. The scrotal skin was red, but not fixed to the tunica vaginalis. No hydrocele was present. Rectal examination showed no abnormality of the prostate or vesicles; but a large mass was felt in the epigastrium, fixed to the posterior abdominal wall. The diagnosis of malignant disease of the testicle with lymphatic dissemination was clear.

The man expressed a great desire to get rid of the tumour, and he was consequently admitted to the wards on May 2, 1925. He then told me of his hæmoptysis, and he coughed up blood each day in hospital. Clinical and radiographic examination showed unmistakable evidence of lung involvement, and he was consequently told that nothing could be done. His brother asked for an unrestricted statement of prognosis, and six months was the period given him. After discussion with the brother, the patient begged me to remove the testicle, as he wished to be rid of the discomfort and pain for what was left of his time. The testicle was accordingly removed through the inguinal canal. He recovered rapidly from his operation, and made preparations for departure to Dublin, where he was going to have deep radiotherapy to the lumbar glands and chest. He was therefore discharged from hospital.

During the last week in June he became suddenly much worse and was re-admitted. It was evident that a fatal issue was but a matter of days, and he died on July 10.

POST-MORTEM.—The examination revealed the following findings :—

*Lungs.*—Both lungs were riddled with metastases, the left lung being worse affected. *Fig. 406* shows a very similar picture, but in my case there was much less normal-looking lung. The left pleural cavity contained a straw-coloured fluid in some quantity, and there was a fibrinous pleurisy, with adhesions at the back at the level of the hilum.

*Heart.*—The superior mediastinum was full of growth which had spread around and enveloped the heart. The muscle and cavities were normal, except for one extremely interesting metastasis. The Eustachian valve was larger than usual and fenestrated, and growing from its margin were several nodules of growth. Many small nodules were present on the contact margins of the tricuspid valve.

*Liver.*—Contained one large secondary nodule, and was the only one in the whole post-mortem examination to give a good histological picture. In all the others necrosis was too advanced. In this nodule the histology is absolutely typical.

*Intestines, Spleen, Kidneys.*—All free of growth.

*Lymphatic System.*—A huge mass of affected glands was found on and between the aorta and superior vena cava about the level of the right renal vessels. Also a small collection on the external iliac vein, and a very definite chain of secondary nodules along the lymphatic running parallel to the vas into the pelvis. Scattered all over the peritoneum of the lateral wall of the pelvis were numerous milk spots of growth connected by a fine white tracery of lymphatics.

**MICROSCOPIC FINDINGS: Testicle.**—Dr. W. D. Newcomb, Pathologist to St. Mary's Hospital, was present at operation, and received the specimen straight from the operating table. Several pieces were immediately taken for special fixation to enable us to stain for glycogen. One half of the specimen was kept intact and put through the usual Kaiserling process. The other half was cut entirely into blocks and fixed in various ways—formol saline, uranium nitrate, formol alcohol, and Ziegl Wallner—and in all thirty-four blocks were prepared and cut.

Apart from the chorionic carcinoma, other teratomatous elements were few and far between, and consisted of a few scattered islands of cartilage and some cysts lined by columnar and by cubical epithelium. The chorionic carcinoma was present in widespread areas. Despite the naked-eye appearance of the tumour, necrosis was not marked, and in only three of the blocks cut did we fail to see this tissue, and in most of them it was present not only in abundance but in such good fixation that it was not easy to settle which field should be selected for drawing. *Figs. 409* and *410* are low- and high-power drawings of easily recognized areas. Masses of vacuolated, granular syncytium are present, and many typical Langhans' cells with mitoses also. *Fig. 408* shows a rather different appearance, a much more orderly arrangement than that seen in *Fig. 409*. Here there does seem to be an attempt to form true chorionic villi. The attempt is not really successful, but the picture is very suggestive. It may be that if anyone is fortunate enough to remove a really early chorionic tumour, such as this in October, 1924, then there will be found typical chorionic villi, and a long step forward towards the proof of Schlagenhanfer's theory will have been taken.

Fig. 411 illustrates the appearance of quite a small area in the tumour. This appears only in one block cut, and in only a corner of that. It is an ordinary adenocarcinoma of a regular arrangement, such as is not often seen in testicular tumours. There is not the slightest evidence to suggest that the cells here lining the acini are anything to do with Langhans' cells, and presumably they are another derivative of the original teratoma.

Only those metastases in the *liver* and the *heart* contained really good histological material. The remainder were all in an advanced stage of necrosis. That in the liver showed a typical arrangement of chorionic tissue as seen in the original tumour. The growth in the heart is of the greatest interest, in that it is composed of numerous villi, being more highly suggestive of true villi than any section I have seen previously. There are also tubules lined by columnar epithelium, representing other teratomatous elements.

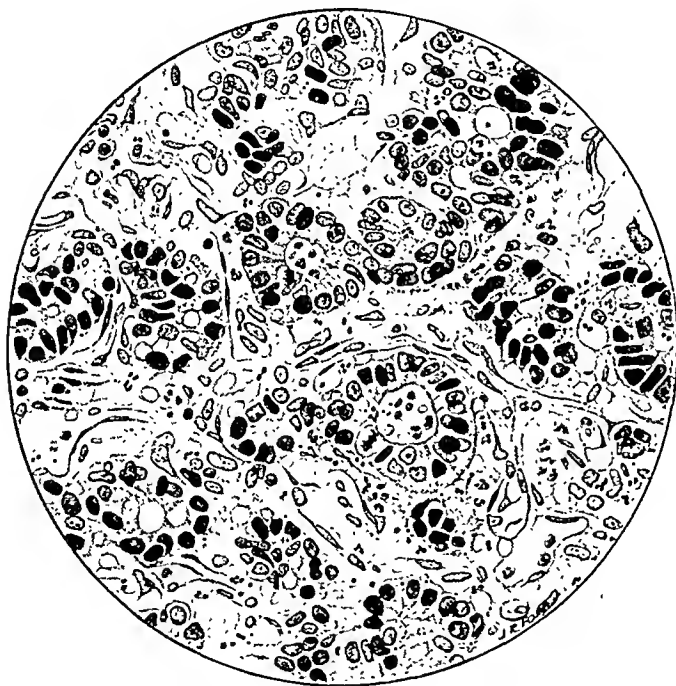


FIG. 411.—A high-power drawing of a small area in the same tumour, showing an adenocarcinoma. The tubulo formation is well seen, and two mitotic figures are present. There are a large number of polymorphonuclear leucocytes scattered throughout the section. ( $\times 300$ .)

In conclusion, it is with great pleasure that I acknowledge my indebtedness to Dr. Newcomb for his help and advice. My thanks are due to Dr. Braxton Hicks, Pathologist to the Westminster Hospital Museum, for access to his material, and his very kind permission to have his specimens drawn. Sir Bernard Spilsbury has most kindly given me a personal communication of a hitherto unpublished case of his own, and Dr. Shore, Curator of the St. Bartholomew's Hospital Museum, has shown me the sections of a tumour in his collection. To them I offer my sincere thanks. And, finally, I am indebted to Mr. Thornton Shiells and Mr. Ford for their admirable drawings. Fig. 407 is a copy of a drawing in Mr. Victor Bonney's paper, and I am greatly indebted to him for his permission to use it.

LIST OF REPORTED CASES OF CHORIONIC CARCINOMA.

Woglom in 1917 brought the list up to 69. I have now further brought this up to date with 40 cases, making 109 in all.

No.	REPORTER	AGE	SIDE	DURATION	RESULT	OTHER TERATOMATOUS ELEMENTS		REMARKS
						Tumour	Metastasis	
1	Malassez and Monod <sup>31</sup>	27	L	Ms. 12	D	No	No	Other teratomatous elements not looked for
2	Carnot and Marie <sup>8</sup>	37	L	10	D	No	No	—
3	Dopter <sup>14</sup>	28	R	6	D	No	No	—
4	Wlassow <sup>75</sup>	—	—	—	—	No	?	No clinical data
5	Wlassow	—	—	—	—	No	?	No clinical data
6	Wlassow	—	—	—	—	Yes	?	No clinical data
7	Wlassow	—	—	—	—	Yes	?	No clinical data
8	Schlagenhauser <sup>39</sup>	43	L	?	D	Yes	No	—
9	Breuss and Schlagenhauser <sup>6</sup>	40	R	2½	D	Yes	No	Interpreted as hydatidiform mole
10	Carey <sup>7</sup>	?	?	?	?	Yes	No	No clinical data
11	Schmorl and Steinert	22	L	9	D	Yes	Yes	—
12	Schmorl <sup>60</sup>	17	?	?	?	Yes	?	—
13	Steinhaus <sup>67</sup>	32	L	6	?	Yes	?	—
14	Risel <sup>57</sup>	20	L	?	?	?	?	—
15	Risel	35	?	?	D	?	Yes	Original growth not examined
16	Emanuel <sup>15</sup>	26	L	4	D	Yes	?	Atypical forms present
17	Westenhoeffer <sup>74</sup>	30	?	?	D	Yes	Yes	—
18	Chevassu <sup>9</sup>	30	L	3	?	No	?	—
19	Hansemann, A. <sup>21</sup>	28	R	?	?	?	Yes	Original tumour not examined
20	Astanazy <sup>1</sup>	24	R	12	D	No	?	Atypical forms present
21	Sternberg <sup>68</sup>	26	R	14	D	No	No	—
22	Scott and Longcope <sup>61</sup>	45	R	2	D	No	No	Undescended testicle
23	Dillmann <sup>13</sup>	32	L	4	D	Yes	No	—
24	Frank, R. T. <sup>19</sup>	40	?	24	?	No	?	—
25	Frank, R. T.	16	L	2	?	Yes	?	Atypical forms present
26	Spilsbury <sup>64</sup>	28	?	2	?	Yes	?	—
27	Hicks <sup>26</sup>	30	R	12	?	Yes	?	—
28	Lawrence <sup>33</sup>	?	?	?	?	Yes	?	—
29	Reinhold <sup>35</sup>	30	R	7	?	Yes	?	—
30	Bernstein <sup>3</sup>	34	L	4	D	No	No	—
31	Bernstein	29	R	?	D	No	No	—
32	Bonney <sup>5</sup>	?	?	?	?	No	?	—
33	Bonney	?	?	?	?	Yes	None	—
34	Orton <sup>47</sup>	38	L	5	D	No	No	—
35	Monckeberg <sup>12</sup>	19	L	10	D	No	No	—
36	Monckeberg	34	R	?	?	No	?	—
37	Monckeberg	Young	?	?	D	Yes	No	—
38	Nicholson <sup>11</sup>	30	R	?	D	?	?	—
39	Nicholson	20	L	9	D	Yes	?	—
40	Nicholson	25	L	24	D	Yes	?	—
41	Chervin <sup>10</sup>	20	R	6	D	No	No	—
42	Jean and Massabuan <sup>23</sup>	44	R	12	?	Yes	?	—
43	Fischer <sup>17</sup>	43	R	?	D	Yes	Yes	Undescended testicle
44	Ohkubo <sup>15</sup>	26	R	12	?	Yes	?	—

LIST OF REPORTED CASES OF CHORIONIC CARCINOMA—continued.

SELECTED CASES OF CHORIONIC CARCINOMA—continued.									
No.	REPORTER	AGE	SIDE	DURATION	RESULT	OTHER TERATOMATOUS ELEMENTS		REMARKS	
						Tumour	Metastasis		
45	Ohkubo .. ..	23		Ms.					
46	Ohkubo .. ..	45	R	?	?	Yes	?	—	
47	Taylor <sup>70</sup> .. ..		R	10	D	?	Yes	Primary not examined	
48	Fink <sup>16</sup> .. ..	28	L	8½	D	Yes	No	—	
49	Klippel and Monier-Vinard <sup>31</sup> .. ..	35	?	?	D	No	?	—	
50	Gruner <sup>22</sup> .. ..	?	?	?	D	Yes	Yes	—	
51	Glaserfield <sup>21</sup> .. ..	25	L	5	?	Yes	?	—	
52	Sigl <sup>62</sup> .. ..	23	L	30	D	Yes	No	—	
		34	R	6½	D	No	No	—	
53	Launois, Masson, and Pinard <sup>32</sup> .. ..	40	L	12	D	?	No	Several congenital abnormalities	
54	Kaufmann <sup>29</sup> .. ..							Primary not examined	
55	Conforti <sup>11</sup> .. ..	43	?	?	D	No	No	—	
56	Zenoni .. ..	46	L	30	D	No	No	—	
57	Marcora <sup>33</sup> .. ..	32	R	?	D	No	No	—	
58	Keenan <sup>30</sup> .. ..	29	L	12	D	Yes	No	—	
59	Hahn <sup>23</sup> (Boestrom) <sup>4</sup> .. ..	20	R	7½	D	No	?	—	
		40	?	7	D	?	No	Epididymis only	
60	Tirumurti <sup>71</sup> .. ..							B.'s case reviewed by Hahn	
61	Frank, A. <sup>18</sup> .. ..	35	R	2	D	No	No	Undescended testicle	
62	Meyer, R. <sup>40</sup> .. ..	30	L	2	?	Yes	?	—	
		23	L	14	D	Yes	No	Primary contained no chorionic epithelioma	
63	Roncali <sup>58</sup> .. ..	18	L	10	D	Yes	?	—	
64	Boncali .. ..	Young	R	?	D	Yes	?	—	
65	Warthin <sup>73</sup> .. ..	35	R	?	D	Yes	No	Hyperplasia of breast	
66	Fritze <sup>20</sup> .. ..	21	L	2	D	Yes	Yes	Metastasis pure chorionic epithelioma, primary none	
67	Cooke <sup>12</sup> .. ..	26	R	?	D	None	None	Hyperplasia of breast	
68	Woglom <sup>76</sup> .. ..	?	?	?	?	Yes	?	No clinical data	
69	Woglom .. ..	35	R	19	D	Yes	No	—	
70	Jackson <sup>27</sup> .. ..	23	R	36	D	Yes	Yes	—	
71	Ooman <sup>16</sup> .. ..	—	—	—	—	—	—	—	
72	Hedinger <sup>23</sup> .. ..	29	R	—	D	Yes	Yes	—	
73	Volkman <sup>72</sup> .. ..	4	R	6	D	Yes	?	No post-mortem allowed	
74	Reckendorf <sup>51</sup> .. ..	24	R	24	D	Yes	Yes	—	
75	Tanner <sup>69</sup> .. ..	—	—	—	—	—	—	—	
76	Richy <sup>56</sup> .. ..	—	—	—	—	—	—	—	
77	Spremolla <sup>65</sup> .. ..	—	—	—	—	—	—	—	
78	Spilsbury <sup>63</sup> .. ..	33	L	?	D	Yes	Yes	Chorionic tissue seen only in metastases	
79	Handfield-Jones .. ..	40	R	7	D	Yes	Yes	—	
80	Handfield-Jones (Spilsbury's case) .. ..	—	—	—	D	Yes	—	—	
81	Handfield-Jones (St. Bartholomew's Hospital Museum) .. ..							—	
82	Dew <sup>82</sup> .. ..	35	L	16	D	Yes	?	Metastasis was not examined	
83-109	Hartmann and Peyron <sup>79</sup> published 27 cases, some of which are not original. New cases not stated.								

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## THE NERVES OF THE STOMACH AND THEIR RELATION TO SURGERY.\*

By E. D'ARCY MCCREA†, MANCHESTER.

*(From the Departments of Anatomy and Physiology, University of Manchester.)*

### INTRODUCTION.

THE advance of physiological knowledge and the increasing connection between physiology and surgery provide the justification for this paper on the stomach and its nerves. Recent advances in the physiology and pathology of these nerves indicate the application of this knowledge to gastric surgery. No attempt has been made to cover the whole field of the literature, and for further details readers are referred to the papers of the authors quoted.

Few will assert that gastric surgery is at the present time in a condition of stability, or rather of comparative stability. As an example, it is only necessary to call attention to the gradual decay of gastrojejunostomy as a routine procedure, first for gastric ulcer and secondly for duodenal ulcer; British surgeons are, however, not so dissatisfied with this operation for juxta-pyloric ulcer as are many surgeons abroad. The marked divergence of opinion with regard to the correct treatment of gastric ulcer is also indicative of an unsatisfactory condition of affairs. Furthermore, surgery fails to relieve or indeed even to treat certain disorders, well known to the clinician and radiologist, which are manifested by gastric signs and symptoms.

For a number of years investigators have endeavoured to determine the relationships existing between the nerves of the stomach and gastric ulcer. The nerves have been studied as a cause of ulcer, as factors productive of the various complications and sequelæ, and as possible sources of cure or prevention. They have also been considered as affording a means whereby surgery may approach the 'nervous dyspepsias'. The writings of Exner, Eppinger and Hess, Bircher, Latarjet, and more recently of Schiassi, are of especial interest in this connection; their work will be referred to in some detail in the following pages.

It may be stated that only two conditions of the stomach and duodenum are submitted to surgical treatment; these are 'peptic' ulcer and neoplasm. Their complications and sequelæ. It is also true that many of the routine operations upon the stomach are mutilating in the extreme, and often out of all proportion to the lesion present, whilst but scant respect is paid to

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† Working for the Medical Research Council.

physiology. Indeed the recent trend appears to be in the direction of more and more mutilating procedures which are only "limited by the powers of recovery of the patient and the resources of technique".

A conclusion reached both from the clinical and radiological standpoints is that, by the present methods, truly satisfactory results are obtained only when definite retention due to organic disease exists. Ryle states that "no attempt should be made to circumvent mechanically a lesion which is producing no considerable mechanical disturbance of function". The suggestion is made that only in definite organic obstruction and in tumour are the results of operation at all justified.

Medicine has covered a wider field than surgery, and the treatment of certain of the alleged non-surgical conditions by means of drugs which act on the nervous mechanism, e.g., atropine, has not been without considerable success. This is, however, often only temporary, and may be associated with undesirable general effects; therefore the hypothesis is put forward that approximately similar results which will be both permanent and local may be obtained by recourse to surgery.

Almost any remarks upon the surgery of the stomach at once lead one into a maze of controversy in which endless side-issues arise to obscure the main discussion. It is my endeavour in this paper to show that operative interference with the nerves of the stomach is both feasible and in certain instances justifiable. It is hoped that the following review of our knowledge of the anatomy and physiology of the nerves, together with some account of their pathology and surgery, may demonstrate the foundations upon which such operations are based and indicate in what directions they may be applied. Those operations which have already been applied to man are described, and operations are suggested by which it is hoped that the conditions referred to under the names of cardiospasm and pylorospasm may be relieved.

### ANATOMY OF THE NERVES.

Modern text-books of anatomy devote but little space to the anatomy of the nerves of the stomach, and are often inaccurate. It is therefore necessary to consider the nerve-supply and its distribution. The anatomy of the vagus and sympathetic supply to the stomach and neighbouring organs has been described in particular by Swan, Kollmann, Perman, Latarjet, and McCrea. The arrangement of the nerve branches has been shown to be of remarkable constancy.

**Vagus.**—It is commonly stated that the left vagus is distributed to the anterior surface of the stomach, the right to the posterior. This is not the case; each vagus sends fibres to both surfaces of the organ. This is evident from the structure of the œsophageal plexus, and from the fact that stimulation of a single vagus in the neck will cause movements of the whole stomach.

The œsophageal plexus is not a meaningless network of nerves, but is a very simple plexus, having a constant basic structure. From it two trunks, anterior and posterior, arise, which may, however, each be represented by two branches. Each vagal trunk contains fibres of both vagi, and I have confirmed this by degeneration methods in the cat. The chief vagal branches

lie along the lesser curvature, but do not reach the pylorus; this receives its nerve-supply from above, from the hepatic branch of the anterior vagal

FIG. 412.—Drawing made from a dissection showing the normal formation and distribution of the anterior vagal trunk. A, Right vagus; B, Left vagus; C, Anterior vagal trunk; E, Hepatic branch; F, Gastric branches; G, Principal anterior nerve of the lesser curvature; H, Branches to pylorus and first stage of the duodenum; P indicates the position of the pylorus, I that of the incisura angularis, and D the diaphragm.

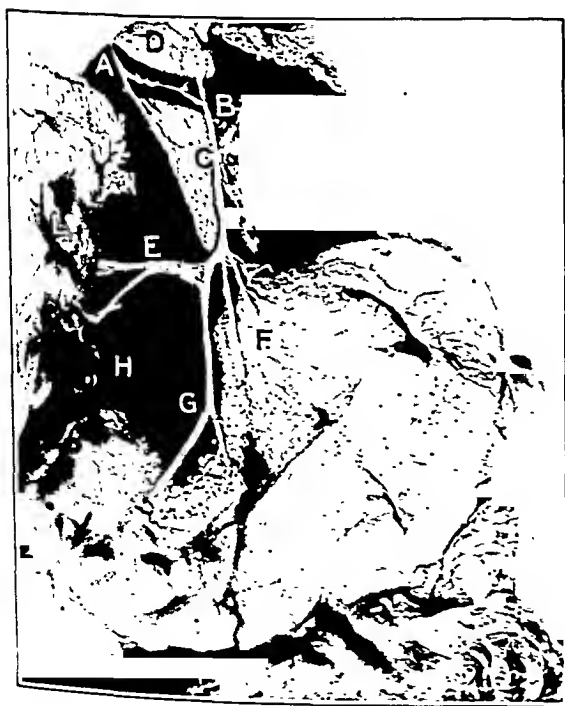
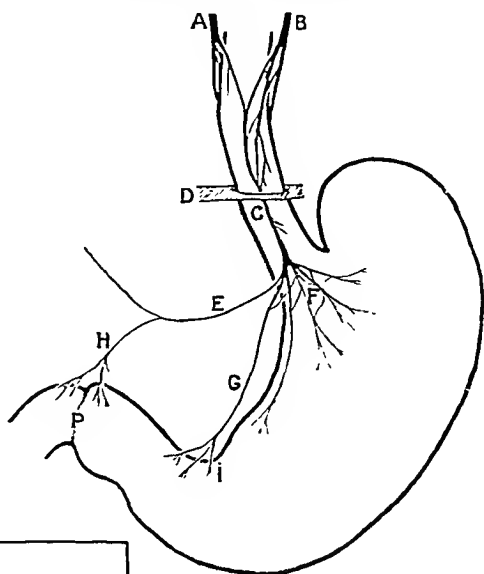


FIG. 413.—Photograph of a dissection illustrating the formation and distribution of the anterior vagal trunk. A, Right vagus; B, Left vagus (anterior branches); C, Anterior vagal trunk; D, Communication from right to left vagus; E, Hepatic branch; F, Gastric branches; G, Principal anterior nerve of the lesser curvature; H, Branch to pylorus and first stage of the duodenum; L is placed on the cut surface of the liver, the left lobe of which has been removed; P indicates the position of the pylorus.

trunk, which crosses high up in the gastrohepatic omentum to reach the porta hepatis. The first stage of the duodenum is supplied in the same manner as the pylorus (Figs. 412, 413).

The posterior vagal trunk supplies a large branch to the cœliac plexus and thus to certain of the abdominal organs, including the pancreas, kidneys, and small intestine. Its gastric division does not reach the pylorus (*Figs. 414, 415*).

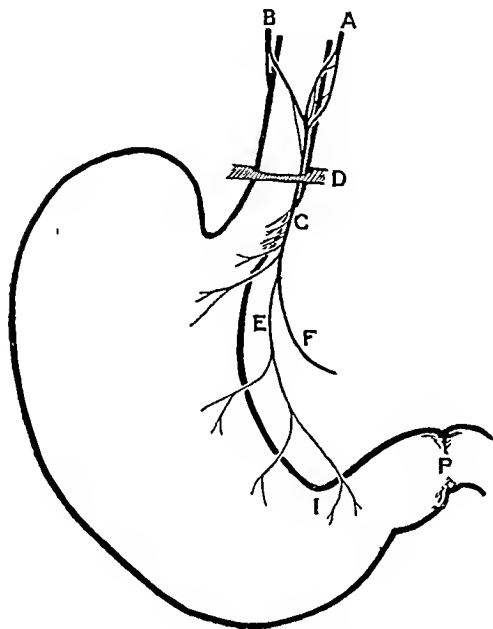


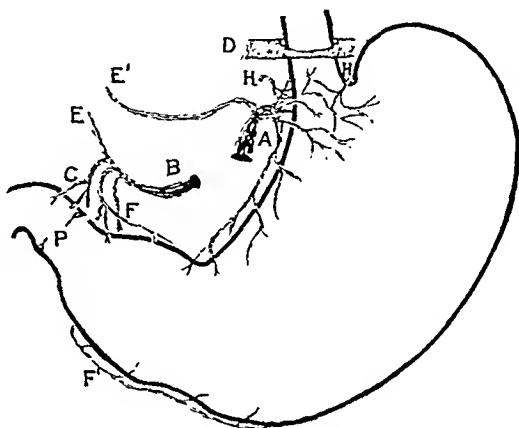
FIG. 414.—Drawing made from a dissection illustrating the normal formation and distribution of the posterior vagal trunk. The stomach has been turned to the right, exposing the posterior surface. A, Right vagus; B, Left vagus; C, Posterior vagal trunk; E, Gastric division, which lies in the coronary falk; F, Celiac division; P indicates the position of the pylorus, I that of the incisura angularis, and D the diaphragm.

FIG. 415. — Photograph of a dissection showing the formation and distribution of the posterior vagal trunk. A, Branches of right vagus; B, Branches of left vagus; C, Posterior vagal trunk; E, Gastric division; F, Celiac division; G, Principal posterior nerve of the lesser curvatur; P indicates the position of the pylorus, and I the site of the incisura angularis.



**Sympathetic.**—The sympathetic fibres are all derived from the splanchnic nerves, and take origin from the cœliac plexus. The majority of the nerve twigs accompany the branches of the cœliac artery, though a few run with the inferior phrenic arteries. Thus the main nerves associate themselves with the left gastric or coronary artery, and are reinforced in the neighbourhood of the cardia by twigs which accompany the inferior phrenic arteries. Other branches run with the hepatic artery, chiefly to the liver, but twigs descend from these to the pylorus and first stage of the duodenum, passing between the layers of the hepatoduodenal ligament and right margin of the gastro-hepatic omentum; some fibres pass with the right gastric or pyloric artery and the right gastro-epiploic artery to the stomach (*Fig. 416*).

**Intrinsic Nerves.**—The intrinsic nervous system of the stomach is usually described as consisting of a nerve net lying between the muscle coats, and of groups of ganglion cells situated either under the serosa or between the muscle bundles. These cells are especially marked in the region of the cardia and in the pars pylorica; their existence in the former situation was first noted by Openchowski. E. Muller has supported the view that such a nerve net is present, and agrees with Dogiel that two types of ganglion cells may be identified; he considers one type to be vagal, the other sympathetic. Kuntz and Abel, on the other hand, believe, as did Gaskell, that the ganglion cells are all vagal in origin; the first-named denies the existence of a nerve net, stating that the myenteric plexus contains only free neurones. In the light of our present knowledge, however, it is impossible to draw very definite conclusions regarding the constitution of the myenteric plexus.



**FIG. 416.**—Drawing to illustrate the manner of distribution of the sympathetic nerves to the stomach. A, Nerves accompanying and distributed with the left gastric or coronary artery (a similar arrangement exists on the posterior surface of the stomach); B, Nerves accompanying the hepatic artery; C, Branches which run a recurrent course in the right free margin of the lesser omentum and with the right gastric or pyloric artery to supply the pars pylorica and duodenum; E, E', Nerves to the liver; F, F', Twigs running with the gastro-duodenal artery and its gastro-epiploic branch; H, H, Twigs with the inferior phrenic arteries. P indicates the position of the pylorus, and D the diaphragm.

### PHYSIOLOGY OF THE NERVES.

The study of the function of these nerves has offered a wide and complicated field for investigation. Certain facts are now well known, largely owing to the work of Langley, Cannon, Elliott, Carlson, and Wheelon and Thomas.

**Vagus.**—The conclusions reached by the majority of investigators, from the results of electrical stimulation of the nerves, is that the vagus

possesses both augmentor and inhibitor actions, but that it is mainly augmentor and accelerator as regards function; it has been emphasized by several workers, however, that the vagus is not a motor nerve in the ordinary sense.

An investigation carried out in association with McSwiney and Stopford showed that the effects obtained chiefly depend upon the pre-existing state of what may be termed the peripheral mechanism of the stomach; thus, the active digesting organ reacts more promptly, the resting one more sluggishly, to stimulation; moreover, in the former the response is primarily inhibitor, while in the latter it is augmentor. In all cases, however, an augmentor response follows stimulation sooner or later, even though the primary response be inhibitor. An inhibitor effect results in cessation of movement, if present, with or without dilatation; the latter effect depends on the presence or absence of an entogastric pressure at the time of stimulation. These results have been recorded by various methods and have been confirmed by direct observation. In addition the X rays have shown that an exaggeration of the normal type of movements occurs, and also that the stomach rapidly empties. It has been shown that 'tonus' changes are limited to the region of the body and fundus, and that the pyloric region takes no part in them (McCrea and McSwiney); Brown and McSwiney also have noted this, and Alvarez has found that a gradient of muscular rhythmicity may be described, most marked proximally, and decreasing as we progress distally towards the pylorus; this, however, Brown and McSwiney could not confirm when working on isolated muscle strips. These observations agree on the whole with the well-known fact that the stomach is functionally divisible into a proximal reservoir and a distal motor mill. Our results show that the vagi regulate both 'tonus' and movement, and, moreover, that these may be independent of one another. Courtade and Guyon, and Carlson, Boyd, and Percy have also noted that the effects obtained on stimulation depend on the existing condition of 'tonus'.

**Sympathetic.**—The splanchnic nerves have been described as having both an augmentor and inhibitor action on the stomach; the chief result of stimulation being inhibition; contraction is said to be more easily obtained at the pylorus than elsewhere. A distinction has been drawn between the type of response obtained on vagal and on splanchnic stimulation. Details are to be found in papers by Morat, Courtade and Guyon, Carlson, Boyd, and Percy, Klee, Wheelon and Thomas, and Carlson and Litt. Carlson suggests that 'tonus' is again responsible for the varying results, hypertonus favouring inhibition and hypotonus contraction. The results of some preliminary experiments carried out with McSwiney show that the type of response is quite dissimilar to that obtained with the vagus; the movements produced occur in the fundus and upper body, and are of the nature of a contraction or relaxation of this region as a whole. The work gives no indication thus far of any antagonism between vagus and splanchnic, save that movement in the active stomach may be inhibited by stimulation of the splanchnics. It would appear that the splanchnics regulate the 'postural tonus' of the stomach, having an augmentor action on the inactive, an inhibitor on the active, stomach.

That the nerves of the stomach contain both excito-secretory and depresso-secretory fibres has been long known from the work of Pavlov and his school, and more recently from that of Suda.

**Central Influence.**—The influence of the central mechanism on the control of the stomach has not been so fully investigated, but again both augmentor and inhibitor effects may be obtained by stimulation of the central cut end of one vagus, provided the other be intact (Morat, Meltzer and Auer, and Carlson, Boyd, and Pearey). The fact that sensory stimulation influences the movements of the stomach is well known; Hughson has recently found, contrary to the usual statement, that emotion hastens the emptying of the stomach. Klee has investigated the vomiting reflex by way of the vagi, and Kronecker and Meltzer have described the receptive relaxation of the cardia on deglutition.

Cannon's theory of the acid control of the pylorus is no longer fully accepted. The functioning of the pyloric sphincter has been especially studied by Wheelon and Thomas, Carlson and Litt, and Alvarez, whilst McCrea and McSwiney have shown that the pyloric region is controlled by the pyloric vagal branch in a manner similar to that in which the remainder of the organ is controlled, but that 'tonus' changes do not occur. The influence of the nerves on the cardia and lower œsophagus appears to run parallel to that on the organ in general.

#### EXPERIMENTAL RESECTION OF NERVES.

The literature on the experimental resection of the extrinsic nerves of the stomach is extensive. The animals most frequently used have been the rabbit, cat, and dog. The site of division varies from the cervical region to the point of entry of the nerves into the stomach wall. Resection of both vagi in the neck has proved fatal, since œsophageal and laryngeal paralysis results, and death follows from respiratory infection; the right nerve may, however, be divided below the origin of its recurrent branch, the animal then usually surviving. Cannon has employed this technique. As we progress distally, the nerves may be resected about the lower part of the œsophagus where they form the œsophageal plexus; Carlson and Watanabe have in dogs employed a transpleural route to reach them here. Another site of division is on the pars abdominalis of the œsophagus; this locality has often been chosen in animals, and Exner has selected it in operations on man. Finally the terminal branches, both vagal and sympathetic, may be severed immediately before they enter the stomach wall. Latarjet has described and performed this operation in man (*see Fig. 419*); he draws attention to three groups of nerves which must be secured: (1) At the cardia; (2) In the right margin of the lesser omentum; and (3) Along the greater curvature, accompanying the right gastro-epiploic artery. He exposes the posterior nerves through an opening in the transverse mesocolon. Bircher has in man divided the vagal branches, three or four in number, high up on the lesser curvature. Stierlin, also in man, has 'circumcised' the stomach in order to entrap all nerves, his incision extending down to the mucous membrane. A somewhat similar operation, though for a different purpose, has been described by Clare.

The splanchnic nerves have been resected intrathoracically in dogs by Watanabe, and Jean has described a technique for this operation in man: they may also be severed immediately after they have passed posterior to the crura of the diaphragm. The latter is the usual site in animals, and here they may be reached either by a transperitoneal or lumbar route. Recently Schiassi has described a procedure designed to divide the pyloroduodenal nerves. The right portion of the lesser omentum is divided in order to secure the sympathetic nerves which descend from above, and the body of the stomach is encircled by an incision which extends through the muscular coat and which severs the parasymphathetic fibres. He has carried out this operation on man.

It is obvious that in all these operations, with the exception of those of Latarjet, Bircher, Stierlin, and Schiassi, fibres destined for other organs as well as those for the stomach and duodenum must necessarily be divided.

Other procedures, which have been carried out in man to intercept the path of the gastric nerves, and to which it is unnecessary to refer in any detail, include: operations on the jugular ganglion of the vagus (Kuttner); division of the posterior nerve roots (Foerster); ganglionectomy (Sicard and Desmarests), and extirpation of the celiac ganglia or plexus (Jaboulay).

**Results of Nerve Section.**—The immediate results of nerve section have been carefully noted by several workers (Klee), but are of but little value from the surgical point of view. They cannot be clearly distinguished from the effects of shock and anaesthesia, and are in part due to nerve irritation. The more remote effects are of greater interest and importance, and in order to present them more clearly *Tables I, II, and III* have been prepared to

*Table I.*—RESULTS OF NERVE SECTION: VAGOTOMY.

AUTHOR	DILATATION	TONUS	CONTRACTIONS		EMPTYING TIME	
			Height or Depth	Interval Between	Initial	Total
Cannon	Within a few days almost normal				Delayed	
Carlson	—	Hypotonus	May appear increased	Increased	—	—
Nieden	Possibly +	A possible hypotonus	No change	No change	A possible slight delay	
Koennecke	+	—	Decreased	Increased	Delayed	Delayed
Watanabe	+	Hypotonus	Decreased	Irregular	Early slight delay	Delayed
Stierlin	+	Hypotonus	Decreased	—	Slight delay	
Litthauer	+ in 9 of 22 cases	Atony in 9 of 22 cases	Diminished motor activity		No change	Delayed
Lichtenbelt	—	—	Increased	—	—	—



Table II.—RESULTS OF NERVE SECTION: SPLANCHNOTOMY.

AUTHOR	DILATATION	TONUS	CONTRACTIONS		EMPTYING TIME	
			Height or Depth	Interval Between	Initial	Total
Cannon	Nil	Nil	Normal	Normal	Very little change	
Carlson	—	Hypertonus	—	Decreased	—	—
Koennecke	—	Hypertonus	Increased	Decreased	Hastened	
Nieden	Nil	No change	No change	No change	No delay	
Watanabe	Slight +	Early hyper-, late hypotonus	Slight increase	—	Almost normal	Hastened
Sticlin	—	Hypertonus	Good		Hastened	
Litthauer	—	—	—	—	No change	Hastened

Table III.—RESULTS OF NERVE SECTION: DENERVATION.

AUTHOR	DILATATION	TONUS	CONTRACTIONS		EMPTYING TIME	
			Height or Depth	Interval Between	Initial	Total
Cannon	Very small stomach	Hypertonus	No change	No change	Slight delay	
Carlson	—	Hypotonus	Increased	Increased	—	—
Nieden	Slight	—	—	—	Slight delay	
Latarjet	+	Hypotonus	Decreased	Increased	Delayed	
Watanabe	+	Hypotonus	Decreased	Increased	Delayed	Delayed

show the results obtained by the chief workers. *Table IV* indicates the methods used.

If we examine these, we find that on certain points complete accord does not exist. However, from the literature to which I have had access the following deductions may be drawn: (1) That section of one vagus or one splanchnic is without effect; (2) That vagal section results in dilatation, diminution of toms, slow and weakened peristalsis, and delayed emptying—in fact, retardation of function; (3) That splanchnic section has almost exactly the reverse effects, and results in an acceleration of function; (4) That sections of all nerves gives similar but slightly less marked effects than vagal section alone. Several observers have stated that these results are not permanent, but that after a period varying from weeks to months, tonus is regained and movements appear normal. Both Watanabe and Koennecke

Table IV.—METHODS EMPLOYED

AUTHOR	ANIMAL	NUMBER	OPERATIONS		
			Vagotomy	Splanchnotomy	Both
Cannon	Cat	—	Cervical, below right recurrent branch	Transperitoneal	Combination
Carlson	Dog	12	Intrathoracic	Transperitoneal	Combination
Latarjet	Dog	—	Denervation		
Koennecke	Dog	8	Abdominal	Transperitoneal	—
	Cat	5			
Watanabe	Dog	20	Intrathoracic	Intrathoracic with division of sympathetic trunk	Combination
Lichtenbelt	Rabbit	—	Abdominal	—	—
	Cat Dog				
Stierlin	Dog	9	Abdominal	(Extirpation of celiac plexus)	—
Litthauer	Dog	22	Cervical Intrathoracic Abdominal	Intrathoracic	—

\* Niesen gives

found that if extirpation of the celiac plexus was substituted for splanchnotomy, the results were more marked and compensation was less complete.

Whether nerve section has any real influence either on secretion or acidity is doubtful. Psychic and reflex secretion disappear on vagal section and therefore the secretory curve is altered. This has been shown by Litthauer, and he also finds that secretion then becomes continuous even in the fasting animal. It appears to be certain that an active juice is still secreted after denervation. The work of Ivy and Whitlow suggests that the second or chemical phase of secretion is dependent on the integrity of the nerves of the pars pylorica; and that this phase is abolished by removal of the antrum, as in partial gastrectomy, has been demonstrated by Dagaew, Lewisohn, and Smidt.

It is necessary to mention some interesting observations made by Ivy on dogs. He produced duodenal ulcers by experimental means, in two series of animals, all of which had been provided with a small stomach, somewhat after the method of Pavlov. The first series possessed a normal nerve-supply to the stomach, but in the second series he carried out resection of both vagi and splanchnics together with extirpation of the celiac plexus. It was found that the presence of an ulcer in the latter series produced less marked alterations in motility and rate of emptying than when the nerves were intact.

## RVE SECTION.\*

METHOD OF EXAMINATION	PERIOD OBSERVED		Notes
	Vagotomy	Splanchnotomy	
; animal tied down	2 weeks to 1 month. +	1 month. +	—
as per œsophagus or by meat gastric fistula	2 weeks to 3 months	2 months	Hunger contractions only studied
X rays	—	—	Results permanent
. before and after opera-	11 months	3 to 4½ months	Division verified post mortem
s and permanent duo- fistula	14 days	1 month	Effects diminished in time; compensation
and Pavlov stomach	—	—	
X rays	(Some weeks to months)		Post-mortems carried out
uent duodenal fistula	(3 weeks at least)		All cervical cases died

: technique

Chronic ulcer formation is a common complication of vagal resection in the rabbit (van Izeren, Ophuls, Greggio, Zironi, 64 per cent). The stomach of this animal, however, functions as a paunch and is never completely empty, whilst experiments with drugs on surviving strips of its musculature give results different from those obtained in the cat and dog (Smith, and Brown and McSwiney). It is not thus comparable to the human stomach, as are those of the cat and dog. In the latter animals gastric ulcer following resection of the vagi is a rarity, according to the majority of investigators; Donati obtained negative results with dogs. Greggio found ulcers in a small percentage of his dogs, and Koennecke in one dog of his series, whilst ulcers are not recorded in the case of the cat. Liechtenbelt, on the other hand, obtained gastric ulcer in 70 per cent of his dogs; he states that for their causation all nerve twigs must be severed, and that they may be prevented if only a fluid diet is given and the stomach washed out daily. Splanchnic section is said to produce acute ulcers and erosions of the stomach and duodenum, but these lesions heal rapidly (Durante, Ivy). Stierlin could not obtain such lesions.

Recovery after these operations and maintenance of good health is the rule if the after-treatment is carried out with reasonable care. Very few out of the many workers have found any difficulty in keeping their animals alive and in good condition. Donati examined the urine of his rabbits after

vagotomy, but found it normal. After extirpation of the cœliac plexus Aldehoff and v. Mering have noted a temporary diarrhoea, and temporary glycosuria, but never the presence of acetone or albumin.

The effects of stimulation of either the vagi or the splanchnic nerves on the small intestine appear to be similar to those obtained with the stomach (Klec); Koennecke, however, finds that vagal resection has little effect on the intestine.

#### THE AUTHOR'S EXPERIMENTS.

The results of a series of experiments which have been carried out with McSwiney and Stopford appear in brief form in *Table V*. The vagi were

*Table V*.—EXPERIMENTAL NERVE SECTION: AUTHOR'S RESULTS.

OPERATION AND NUMBER	DILATATION	CONTRACTIONS		EMPTYING TIME	
		Depth	Interval Between	Initial	Total
Vagotomy (17)	None detected	Increased	Decreased or no change observed	Marked decrease	Slight decrease or within normal limits
Splanchnotomy (4)	None	Increased	No change detected	Marked decrease	Decrease
Vagotomy and splanchnotomy (4)	None detected	Increased	No change detected	Marked decrease	Within normal limits
Unilateral vagotomy (11)	No variations from within the normal limits detected				

divided on the pars abdominalis of the œsophagus, and the splanchnics immediately below the crura of the diaphragm, where they were reached by the abdominal route. Partial resections have in addition been carried out, the hepatic branch of the vagus has alone been divided, vagotomy exclusive of this nerve has been performed, and complete denervation of the pylorus has also been effected. When one vagus was to be severed, this operation was performed in the cervical region. The results were noted by means of repeated X-ray examinations, carried out at intervals after operation, and compared with similar examinations made before operation. Observations have been made over periods ranging up to nine months with double vagotomy, seven months with double splanchnotomy, eight months with denervation, and twenty-two months with unilateral vagotomy. To summarize these findings:—

1. The results of vagotomy or complete denervation, which are almost identical, may each be divided into two groups—those which are temporary or which are compensated for in time, and those which may be considered permanent. The first group comprises paresis and dilatation; the second includes increased depth of peristalsis, possibly only apparent, some slight dilatation, and a marked decrease in the initial, with a slight decrease in the

total, emptying times. The early initial emptying is the most prominent feature of these, the food appearing to pour into the duodenum, perhaps

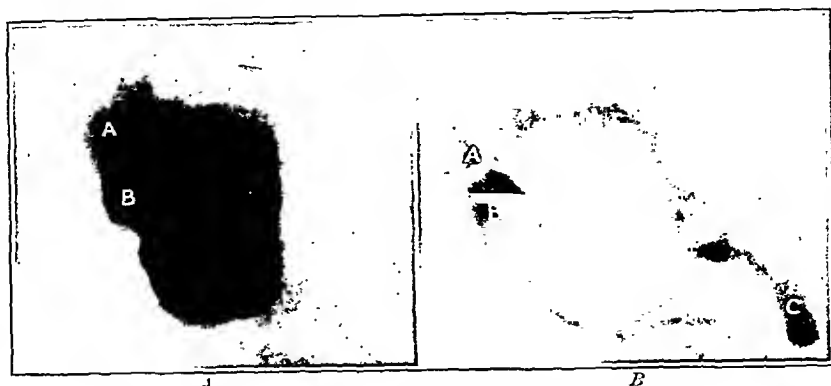


FIG. 417.—Dog 5. Double vagotomy. Plates taken eight months after operation.

A: Five minutes after barium meal, food leaving stomach. A, Duodenal cap; B, Pyloric antrum.

B: Ten minutes after meal. A, Duodenal cap; B, Pyloric antrum; C, Cæcum.

Before operation no food left the stomach within twenty-five minutes after feeding. Total emptying time reduced by approximately one hour. Peristalsis appears normal.

unassociated, at least at first, with peristalsis. This appears to be due to a semipatulous condition of the pyloric sphincter. In a few instances a terminal delay was noted after a very rapid preliminary emptying. (Fig. 417.)

2. Splanchnotomy results in increased depth of peristalsis, and the stomach appears small and tubular in form. These effects, again, gradually become less marked, and the organ returns towards the normal state. In this instance also the initial emptying time remains markedly shortened, while there is an appreciable decrease in the total emptying time. The rapid emptying seems to depend rather on increased activity than on a patulous condition of the sphincter. (Fig. 418.)

3. Denervation of the pylorus or resection of the vagal branch to the pylorus gives similar results to vagotomy or complete denervation of the stomach, but the early paresis and dilatation do not appear.



FIG. 418.—Dog 5. Double splanchnotomy. Five months after operation. Plate taken five hours after a barium meal, showing acceleration of gastric and intestinal function; a considerable portion of the meal is in the large gut. A, Stomach; B, Small gut; C, Large gut; D, Rectum. Before operation the total emptying time was invariably more than seven hours.

primary, even though morphologically or at least macroscopically unrecognizable, injury in a root region of the vagus, or through an anomaly of the nerve regions included in the reflex arcs, a quite remote, severe circulatory disturbance leading to tissue necrosis—e.g., in the gastric and duodenal wall—may be produced". Whilst not entirely subscribing to this view as supplying the explanation of the cause of all chronic ulcers, we must admit that a certain proportion may thus be produced; and it would appear that a lesion of the gastric or duodenal wall, the result of infection or injury, is more likely to become chronic if an irritation of the nerves is set up. In this relation it is of interest to note that all chronic gastric ulcers are situated in the region of the lesser curvature, the so-called 'ulcer-bearing' area, and that this site corresponds to the area through which the nerves enter the stomach wall. Askanazy and also Dible have found that quite prominent nerves are often to be seen in the floor of an ulcer and in the surrounding fibrosis, and in addition that ganglion cells of the myenteric plexus may be found. This nerve irritation results in local spasm and ischaemia, and thus prepares a suitable soil for autodigestion. There is no doubt, moreover, that distant reflex disturbances and spasm may be set up by such irritation, and several observers have shown that these occur with ulcer (Klee, Borchers, Stierlin, Loeper and Forestier, Saloz and Moppert, and Held and Roemer). From the anatomy of the nerves it is easy to explain thus the occurrence of pylorospasm in ulcer of the lesser curvature, and of gastrospasm in duodenal ulcer, both of which are common radiological findings. Woodburn Morison\* states that ulcer of the lesser curvature usually retards emptying, and that in pyloric ulcer retardation is the rule, whilst in duodenal ulcer acceleration is the common occurrence, often, however, associated with a terminal delay. He finds that in the 'nervous dyspepsias' any of these findings may be mimicked, and in this he differs from Saloz and Gilbert, who find but little variation from the normal in these patients. In the case of pyloric ulcer the site and local irritation serve to explain the retention, and in other situations any alteration may be explained by a reference to the nerves, whether they are involved or not, and whether, if involved, the lesions are irritative or destructive. Askanazy has classified the changes undergone by the nerves in the base of an ulcer into (1) destructive, (2) passive, which include peri- and endoneuritis, and (3) productive, the last including neuroma formation. Reflex spasms—the result of ulcer—especially pylorospasm, cause retention; retention and infection favour the further development of an ulcer, and so a vicious circle is set up. Alternatively it is clear that reflex pylorospasm, however caused, results in retention and hyperchlorhydria (Bolton), thus paving the way for the development of ulcer.

Lim, Ivy, and McCarthy assert that gastric secretion is in part due to mechanical stimulation, i.e., to distention; they believe that this explains to some extent the 'hypernormal gastric secretion' in cases of pyloric obstruction. Pylorospasm has therefore a dual effect on secretion and acidity, in that, first, increased secretion is promoted, and, secondly, the normal neutralization by reflux through the pylorus is prevented. It would thus

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\* Personal communication.

appear that a lesion in any part of the course of the nerve paths is a factor in the development of gastric ulcer. The lesion may be primary and situated at a distance from the stomach, thus fulfilling the conditions of the 'neurogenic theory', or may be secondary and either intra- or extra-gastric, the result of inflammation or injury of the stomach wall or of a more remote part such as the appendix.

It has been shown that if the pyloric antrum is completely removed, as in partial gastrectomy, there occurs a loss of the chemical or second phase of gastric secretion, achlorhydria frequently resulting (Dagaew, Lewisohn, Smidt). Berg therefore recommends such a procedure in all cases of ulcer, for with regard to acidity the results of gastrojejunostomy vary, and it is an accepted fact that chronic gastric ulcer can only occur in the presence of hydrochloric acid (Mann and Williamson); he has observed that a simple pylorectomy does not produce this result. The work of Ivy and Whitlow suggests that the nerves of the pars pylorica play a great part in this second phase of secretion, and that their section abolishes it. Their work has failed to confirm that of Edkins and Tweedy, who described 'gastric secretin' or 'gastrin'.

The radiological findings which have been made in certain cases of hour-glass stomach are of interest. In a few instances the proximal pouch empties freely and rapidly, yet delay occurs in the distal pouch because of pylorospasm. Woodburn Morison\* finds that such pylorospasm is frequent in patients with hour-glass stomach. It is without doubt reflex in origin, and again explained by the distribution of the nerves, if we except those instances, not very infrequent, in which a second lesion causing local spasm is present near the pylorus. The type in which a rapidly emptying proximal pouch is associated with pylorospasm affords an explanation of the good results which have been recorded in a few cases when gastrojejunostomy to the distal pouch has been performed.

**'Nervous Dyspepsias'.**—The conditions known by the title of the 'gastric neuroses' or 'nervous dyspepsias' must now be considered. They may be divided into two broad classes, the vagotonic and the sympatheticotonic of Eppinger and Hess; the first or vagotonic group show signs and symptoms indicative of increased vagal action, the second of increased sympathetic action. Excessive vagal control is evidenced by hypermotility, either with or without 'hypertonus', as shown by spasm; increased sympathetic control results in atony and 'hypotonus'. The increase in vagal action may be real or apparent; in the latter instance there is lack of sympathetic control, and the resultant unbalanced action appears in the form of excessive vagal influence; similarly increased sympathetic action may also be real or apparent. Many authors believe that the sympathetic plays a greater part than the vagus in these disorders, and Renard has described a definite "syndrome sympathique général".

These conditions have at present no known pathology, but are recognized and described from their signs and symptoms. In none have any lesions been demonstrated either in the stomach or its nerves; there is, however,

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\* Personal communication.

4. The effects of resection of the nerves on the function of the small intestine are similar to, but less marked than, those occurring in the stomach.

It is obvious that our findings in vagotomy and denervation, and the conclusions drawn from the literature, differ most markedly on the question of emptying times. The results of splanchnotomy are similar to those previously recorded. Every precaution was taken in this series to prevent the possibility of psychic inhibition before operation, to establish the normal variations in the individuals examined, to employ only the X rays without other procedure in noting the results, and to confirm the operations post mortem.

The pylorospasm and cardiospasm which have been described as immediate on vagal section (Klee, Exner) are purely temporary, and are no doubt due to the irritation of the nerve section. The permanent delay in emptying time which has also been noted may be attributed to the escape of the pyloric nerves at operation, which can occur either in the Latarjet operation or in subdiaphragmatic resection of the nerves. The early gastric paresis then contributes to the retention, and is probably aided by reflex irritation of the remaining fibres. This explanation undoubtedly fitted the facts in the case of one dog of this series in which an incomplete Latarjet operation was carried out. It must be recalled, however, that in a few instances a terminal delay was observed; this occurred in the dog only, and followed complete denervation. The control of the emptying time by X-ray examination is more reliable than by means of a duodenal fistula which several workers have employed. Furthermore, it would appear that insufficient attention has been paid to the individual variations of the normal, and in many instances the time of observation has been too short, not allowing repeated examinations at sufficient intervals. Hughson's observations on dogs, that any abdominal operation—even a simple laparotomy—delays the emptying of the stomach, is of importance.

Ulcer formation has been observed in this series in approximately 50 per cent of rabbits after vagotomy, but not in cats or dogs. Following splanchnotomy, multiple duodenal erosions have been found in one cat only. No histological changes were observed in any of the abdominal organs examined post mortem, and the urines of the dogs examined qualitatively after operation were normal; the animals maintained their weight satisfactorily. Recovery is rapid, but some care, especially as regards feeding, is necessary for the first week or ten days. A temporary constipation after resection of the vagi, and a temporary diarrhoea after division of the splanchnics, is often observed. In general, after these nerve resections the great majority of the animals appeared to live normal lives, and no mortality that was attributable to the nerve sections occurred, if rabbits are excepted.

Brown has examined the musculature of the stomach of a few of the operated animals. His results up to the present show that after vagotomy the rate of contraction of the pyloric sphincter is slowed, while that of the body is increased. After section of both splanchnic nerves the rhythm of the pyloric sphincter is increased, but there is no change, beyond the limits of the normal, in the remainder of the stomach. Complete denervation results in a slightly lowered rhythm of the pyloric sphincter, while that of the antrum and body



is somewhat increased. His findings therefore run parallel to those observed with the X rays.

**Conclusions in Regard to the Physiology of the Nerves.**—The general conclusions which may be drawn with regard to the functions of the extrinsic nerves of the stomach, both from the literature and from the work with which I have been associated, are as follows:—

The vagi and the sympathetic nerves may each act either as augmentors or inhibitors of the stomach. The vagus may be described as predominantly augmentor. It is not suggested that one is definitely antagonistic to the other; this is not the case; they belong to different systems, or rather to different parts of the same system, and govern different functions. The sympathetic is concerned chiefly with the 'postural tonus' of the organ, the vagus both with this and with the motor function. Each nerve is undoubtedly both efferent and afferent, transmitting centrifugal and centripetal impulses, and concerned with the motor, vasomotor, secretory, and sensory functions of the organ. They act as regulators of the stomach, and normally guide it to function in harmony with other parts of the body by means of the reflexes passing through them. One vagus or one splanchnic may be divided without influencing the organ in any way; the remaining colleague of either then suffices to carry on the work of both. It would appear that both vagi and splanchnics are together necessary for the normal regulation of the stomach, for when both vagi are severed effects appear which are due either to the abolition of vagal impulses, with an associated exaggeration of splanchnic action, or to one of these alone; such results, however, are temporary, and gradually diminish. If both splanchnics are divided, comparable effects occur, presumably of similar origin; there is, however, a more definite suggestion of vagus over-action in this case than of splanchnic over-action in the preceding instance, and it is also evident for a longer period. The cutting out of one system of regulators therefore gradually results in a diminution and in almost a disappearance of control by the remaining system, the final result approaching but not attaining the effects of section of both. In this complete denervation, the intrinsic automatic mechanism takes on the proper functioning of the stomach no longer guided by external reflexes.

As regards secretion after nerve section, two facts only are definitely stated: (1) On section of the vagi 'psychic secretion' is lost: and (2) An active gastric juice is still secreted. It is probable that division of the nerves of the pyloric antrum abolishes the chemical or second phase of secretion. Further work on the question of secretory changes following nerve resection is necessary, and it is possible that certain of the motor effects obtained are in part dependent on changes in acidity and secretion brought about by the denervation.

### THE NERVES IN RELATION TO DISEASE.

**Chronic Gastric Ulcer.**—The part played by the nerves in the study of disease of the stomach is of considerable interest and importance. The neurogenic theory of causation of chronic gastric ulcer must first be referred to. This theory is attributed to Rössle, and is supported by many authors, including von Bergmann; Gruber states that it is held "that through a

primary, even though morphologically or at least macroscopically unrecognizable, injury in a root region of the vagus, or through an anomaly of the nerve regions included in the reflex arcs, a quite remote, severe circulatory disturbance leading to tissue necrosis—e.g., in the gastric and duodenal wall—may be produced". Whilst not entirely subscribing to this view as supplying the explanation of the cause of all chronic ulcers, we must admit that a certain proportion may thus be produced; and it would appear that a lesion of the gastric or duodenal wall, the result of infection or injury, is more likely to become chronic if an irritation of the nerves is set up. In this relation it is of interest to note that all chronic gastric ulcers are situated in the region of the lesser curvature, the so-called 'ulcer-bearing' area, and that this site corresponds to the area through which the nerves enter the stomach wall. Askanazy and also Dible have found that quite prominent nerves are often to be seen in the floor of an ulcer and in the surrounding fibrosis, and in addition that ganglion cells of the myenteric plexus may be found. This nerve irritation results in local spasm and ischaemia, and thus prepares a suitable soil for autodigestion. There is no doubt, moreover, that distant reflex disturbances and spasm may be set up by such irritation, and several observers have shown that these occur with ulcer (Klee, Borchers, Stierlin, Loeper and Forestier, Saloz and Moppert, and Held and Roemer). From the anatomy of the nerves it is easy to explain thus the occurrence of pylorospasm in ulcer of the lesser curvature, and of gastrosplasm in duodenal ulcer, both of which are common radiological findings. Woodburn Morison\* states that ulcer of the lesser curvature usually retards emptying, and that in pyloric ulcer retardation is the rule, whilst in duodenal ulcer acceleration is the common occurrence, often, however, associated with a terminal delay. He finds that in the 'nervous dyspepsias' any of these findings may be mimicked, and in this he differs from Saloz and Gilbert, who find but little variation from the normal in these patients. In the case of pyloric ulcer the site and local irritation serve to explain the retention, and in other situations any alteration may be explained by a reference to the nerves, whether they are involved or not, and whether, if involved, the lesions are irritative or destructive. Askanazy has classified the changes undergone by the nerves in the base of an ulcer into (1) destructive, (2) passive, which include peri- and endoneuritis, and (3) productive, the last including neuroma formation. Reflex spasms—the result of ulcer—especially pylorospasm, cause retention; retention and infection favour the further development of an ulcer, and so a vicious circle is set up. Alternatively it is clear that reflex pylorospasm, however caused, results in retention and hyperchlorhydria (Bolton), thus paving the way for the development of ulcer.

Lim, Ivy, and McCarthy assert that gastric secretion is in part due to mechanical stimulation, i.e., to distention; they believe that this explains to some extent the 'hypernormal gastric secretion' in cases of pyloric obstruction. Pylorospasm has therefore a dual effect on secretion and acidity, in that, first, increased secretion is promoted, and, secondly, the normal neutralization by reflux through the pylorus is prevented. It would thus

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\* Personal communication.

appear that a lesion in any part of the course of the nerve paths is a factor in the development of gastric ulcer. The lesion may be primary and situated at a distance from the stomach, thus fulfilling the conditions of the 'neurogenic theory', or may be secondary and either intra- or extra-gastric, the result of inflammation or injury of the stomach wall or of a more remote part such as the appendix.

It has been shown that if the pyloric antrum is completely removed, as in partial gastrectomy, there occurs a loss of the chemical or second phase of gastric secretion, achlorhydria frequently resulting (Dagaew, Lewisohn, Smidt). Berg therefore recommends such a procedure in all cases of ulcer, for with regard to acidity the results of gastrojejunostomy vary, and it is an accepted fact that chronic gastric ulcer can only occur in the presence of hydrochloric acid (Mann and Williamson); he has observed that a simple pylorotomy does not produce this result. The work of Ivy and Whitlow suggests that the nerves of the pars pylorica play a great part in this second phase of secretion, and that their section abolishes it. Their work has failed to confirm that of Edkins and Tweedy, who described 'gastric secretin' or 'gastrin'.

The radiological findings which have been made in certain cases of hour-glass stomach are of interest. In a few instances the proximal pouch empties freely and rapidly, yet delay occurs in the distal pouch because of pylorospasm. Woodburn Morison\* finds that such pylorospasm is frequent in patients with hour-glass stomach. It is without doubt reflex in origin, and again explained by the distribution of the nerves, if we except those instances, not very infrequent, in which a second lesion causing local spasm is present near the pylorus. The type in which a rapidly emptying proximal pouch is associated with pylorospasm affords an explanation of the good results which have been recorded in a few cases when gastrojejunostomy to the distal pouch has been performed.

**'Nervous Dyspepsias'.**—The conditions known by the title of the 'gastric neuroses' or 'nervous dyspepsias' must now be considered. They may be divided into two broad classes, the vagotonic and the sympatheticotonic of Eppinger and Hess; the first or vagotonic group show signs and symptoms indicative of increased vagal action, the second of increased sympathetic action. Excessive vagal control is evidenced by hypermotility, either with or without 'hypertonus', as shown by spasm; increased sympathetic control results in atony and 'hypotonus'. The increase in vagal action may be real or apparent; in the latter instance there is lack of sympathetic control, and the resultant unbalanced action appears in the form of excessive vagal influence; similarly increased sympathetic action may also be real or apparent. Many authors believe that the sympathetic plays a greater part than the vagus in these disorders, and Renard has described a definite "syndrome sympathique général".

These conditions have at present no known pathology, but are recognized and described from their signs and symptoms. In none have any lesions been demonstrated either in the stomach or its nerves; there is, however,

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\* Personal communication.

one disease in which, when gastric symptoms of hypermotility and spasm occur, lesions may be found in the vagus and in the posterior nerve-roots and ganglia—I refer to the gastric crises of tabes dorsalis. Furthermore, that the nerves of the stomach are not immune to pathological changes is obvious from various case reports (Longet, Massary and Walser).

Eppinger and Hess believe that unbalanced action of the nerves may be constitutional in origin, and they discuss the possibility of endocrine instability as a cause, calling attention to the findings in Addison's disease and lymphatism; furthermore, they have shown that vagotonia may be generalized or local. Hernando also emphasizes the action of the endocrine organs upon the stomach, both directly through their hormones and by the action of these on the autonomic system. Latarjet believes that the source of the disturbance may be either central or peripheral, and classifies his cases into psychopathies and gastropathies. In Ryle's classification of the dyspepsias these cases are included in *Groups II* and *IV*, the former being the nervous or psychogenic and the latter the irritative; he divides the symptom complexes into pressor and depressor, which appear to correspond in general to the vagotonic and sympatricotonic. Gastrospasm, including cardiospasm and pylorospasm, has been studied from the clinical and radiological aspects by Held and Roemer.

Amongst the symptom complexes are some which have received generally accepted titles, because the clinical findings in them are definite and constant; such are hyperkinesis or supermotility, atony, incontinence of the pylorus, insufficiency of the cardia, and with these must be included cardiospasm (idiopathic dilatation of the œsophagus) and pylorospasm. Whether the so-called hypertrophic pyloric stenosis of infants should be added to these is not definite. I am not aware that this condition has as yet been satisfactorily distinguished from pylorospasm. A contracted, tubular condition of the pars pylorica is not uncommon at post-mortems on infants, but is rare in adults; this would seem to point to an increased irritability of the nerves of the infant. Should either a lowered threshold to stimuli or an increased excitability of the nerves be present, motor disturbances would be expected to be of frequent occurrence, and, in the instance of pylorospasm, an explanation would be supplied. It is possible that pylorospasm and hypertrophic pyloric stenosis of infants may be but degrees of the one condition; Cameron tends towards this view, and in a recent review of the findings in congenital pyloric stenosis notes that the obstruction is invariably intermittent and that no true stenosis exists.

The theories advanced to explain congenital hypertrophic stenosis may be applied with equal force to all forms of sphincteric obstruction in which no organic causes are found. There seems no sound reason why the presence or absence of muscular hypertrophy should be selected as a definite line of cleavage marking off 'stenosis' and 'spasm' into distinct entities; it is quite conceivable that spasm may result in hypertrophy (see also Held and Roemer). Theories which have found most favour in the explanation of these conditions are those of spasm, muscular inco-ordination, and achalasia, and it may be presumed that in any given instance of spasm or paralysis the cause of either is extrinsic or intrinsic in origin. An intrinsic source is best illustrated by

the presence of some local, demonstrable lesion; an extrinsic, which acts through the nerves, has been shown above to be equally efficacious, and Cameron suggests, when referring to such conditions in infants, that they may be due to an unequal development of the nervous augmentor and inhibitor apparatus. In the absence of a demonstrable intrinsic lesion it is necessary to seek for an extrinsic source. An extensive literature on "spastic tumour of the pyloric canal" will be found in a paper by Lerche.

Secretory and sensory gastric neuroses are part and parcel of the conditions referred to above; in them these symptoms predominate, but there are always associated motor disturbances; hyperchlorhydria may be instanced as an example. A careful review of the functional achylia has been made by Udaondo.

The accompanying brief case-notes illustrate the two chief types of the 'nervous dyspepsias'.

*Case 1.—Vagotonic or pressor type.*

A. B., age 45. Male. Intermittent attacks of indigestion extending over a period of three years.

X-ray examination showed stomach lying somewhat low, no filling defect, pylorus patent, duodenal cap well formed, peristalsis active, emptying with great rapidity but a final delay.

OPERATION.—No sign of gastric or duodenal ulcer. Appendix removed.

Two and a half years later no improvement in general condition. Still suffers from indigestion.

*Case 2.—Sympatheticotonic or depressor type.*

M. N., age 52. Male. History of twenty years' indigestion.

Bismuth meal showed a large atonic type of stomach. Pylorus opposite 3rd lumbar vertebra. Five-hour bismuth meal: Delay in emptying.

Clinically: Atonic dyspepsia.

Radiographically: Atonic stomach with no evidence of gastric or duodenal ulcer.

OPERATION.—No ulcer; no growth.

We may conclude that disorders of the gastric function can be produced either by hypo- or hyperfunction of the extrinsic nerves. The primary cause which gives rise to such disordered function may be located: (1) Peripherally; (2) In the nerve paths; (3) Centrally.

1. Irritation, the result of inflammation, is no doubt the most common peripherally situated cause. The irritation may have its origin at a distance, e.g., the appendix or gall-bladder, and thus produce a reflex dyspepsia which may be termed of remote origin, in contradistinction to that which lies in the wall of the stomach or duodenum and may be referred to as proximal. Pylorospasm, supermotility, and hyperchlorhydria are all easily explained on this basis; in addition, it must be noted that factors favourable to the development of ulcer may be thus brought into being; indeed, certain conditions may well be termed pre-ulcerous.

2. Lesions in the nerve paths are exemplified by the findings in the gastric crises of tabes dorsalis; lesions of the posterior nerve-roots which result in gastric disturbance are named 'gastro-radicularitis' by Bouchut and Lanny.

3. Disorders of central origin may be constitutional, as suggested by Eppinger and Hess, and possibly a result of endocrine instability.. It is very probable that the number of 'neuroses' will be materially reduced by the discovery of lesions and inflammations of the peripheral mechanism and nerve paths which research has not as yet demonstrated. The part which may be played by toxæmia and by drugs in their causation must not be lost sight of.

Irritation on the one hand, and diminished function on the other, with various combinations or stages of these, may thus account for a number of the 'nervous dyspepsias', of which even the differential diagnosis is now very incomplete.

It is stated that disorders of peristalsis may arise from extensive scarring, or be due to a local removal of the stomach wall, especially if from the region of the lesser curvature. Alvarez has raised this question, and suggests that a sleeve resection is preferable to a wedge; Berg also states that disturbances of motility follow the wedge resection; he believes that a tract of conducting tissue may pass down the lesser curvature and connect the nodal tissue described by Keith in the region of the cardia with another node near the incisura angularis. That a node may exist in this region is suggested to him by the observation that the movements of the cardiac and pyloric portions of the stomach are quite distinct and may occur separately (Berg and Crohn, McCrea, McSwiney, Morison, and Stopford, and McCrea and McSwiney). Cannon has shown that complete circular incisions of the tunica muscularis do not subsequently interfere with peristalsis. It is of interest to note in this connection that Stierlin ascribes better results to the sleeve resection because in this operation all the nerves must necessarily be divided.

### THE NERVES IN RELATION TO SURGERY.

The nerves of the human stomach have received considerably more attention at the hands of the surgeon than is perhaps generally known. I believe that Jaboulay in 1899 was the first to advocate surgical interference with them, the operation he suggested being extirpation of the celiac plexus. Mingazzini in the same year performed the first intradural division of the posterior nerve-roots for a case of gastric crises. I desire in particular to refer to the work of Exner, 1911, of Bircher, 1920, of Latarjet, 1921, and of Schiassi, 1925, and of those who have followed their techniques (*Fig. 419*).

Exner performed subdiaphragmatic resection of the vagus, and his cases were all tabetics with gastric crises. He considered that the vagus controlled vomiting, and that Foerster's operation was only of use in cutting off sensation via the splanchnics. He observed on section of the nerves an immediate dilatation, atony, and pylorospasm, and because of the last added to his procedure a gastrojejunostomy or a gastrostomy. *Table VI* is from Exner and Schwarzmann, 1914-15. I have added a case published by Thomsen.

Bircher's operation would appear to be an incomplete resection of the vagi. He has operated on 20 cases which clinically resembled gastric ulcer: one proved to be an ulcer, in 6 the diagnosis was doubtful even at operation, and in the remainder no ulcer was present. Before operation almost all these cases showed an atonic stomach. He states that the results were good;

vomiting, nausea, and belching were abolished, pain disappeared, and secretion and acidity were reduced, the latter very considerably. Subsequent X-ray examination showed an increase of tonus.

Latarjet's operation, to the technique of which I have referred, is equivalent to section of all the extrinsic nerves. The results in man, he finds,

FIG. 419.—Photograph of a dissection of the nerves of the stomach on which are illustrated the sites of division of the nerves in the operations of Latarjet, Exner, Stierlin, and Schiassi. V, Anterior vagal trunk (two divisions); J, Gastric branches; K, Gastrophrenic branches; L, Liver; M, Pyloric and duodenal nerves; N, Principal anterior nerve of the lesser curvature; A-B, The Latarjet operation (three steps; the fourth, in which the nerves on the posterior surface are reached, is not indicated); C-D, Exner's operation; E-F, Stierlin's operation; G-H, Schiassi's operation (two steps).



Table I'1.—VAGOTOMY FOR GASTRIC CRISIS. (Exner and Schwartzmann, 1915.)

AUTHOR	NUMBER OF CASES	CURE	NO RESULT	OBSERVATION TOO SHORT	IMPROVED	WORSE	DEATHS
Exner ..	14	7	2	2	2	0	1
Ranzi ..	3	1	1	1	—	—	—
Kuttner ..	1	—	—	—	—	1	—
Hildebrand	1	1	—	—	—	—	—
Neumann ..	1	1	—	—	—	—	—
Total ..	20	10	3	3	2	1	1
Thomsen ..	1	1	—	—	—	—	—

are exactly similar to those described by him in dogs. His aim was to cut off sensation, diminish the frequency and intensity of the contractions, and probably to diminish acidity. I have found in all 59 cases operated on by Latarjet, Wertheimer, Pauchet, and Gianolla; the details are not fully given, but only in 18 was a simple denervation without other procedure carried out (*Table VII*). Their results over a comparatively short observation period are good. Considerable pain is said to follow the operation for the first twenty-four hours, but is easily controlled by morphia. Their cases include examples of gastric ulcer, of the gastric crises of tabes, and of certain of the stomach neuroses, which Latarjet classifies as gastropathies; he states that those most suitable for operation are the vagotonics who show pain, hypertonus, hypermotility, and vomiting, and in whom no ulcer is present. However, even the psychopathies are improved.

I have found no record of any case operated on by the technique described by Jean.

*Table VII.*—DENERVATION WITHOUT OTHER PROCEDURE. (*Latarjet, 1921-3.*)

GASTROPATHY	NUMBER OF CASES	RESULT	NOTES
Vagotonic	1	Disappearance of all symptoms	A previous exploration and appendicectomy
Vago-sympathetic	2	Cure	Observation up to 18 months
Vago-sympathetic with ptosis	5	Cure	Observed 2 to 6 months. Operation by Pauchet in 2 of these
Vago-sympathetic with dilatation	1	Cure for 1 year, then slight recurrence	
Vago-sympathetic with hypotonus	1	Cure	Observed 2 months
Unclassified	4	Cure	Observation up to 1 year
Gastric crises	4	Very marked improvement in pain and vomiting	—

Steinthal operated on two cases of gastric ulcer by the method of Stierlin. The first showed atony, hypersecretion, and hyperacidity; after operation peristalsis was found to be normal, but the hypersecretion was unaltered. The second showed increased peristalsis, a six-hour residue, hypersecretion; after operation increased peristalsis was still present, secretion was further increased. He observes that the operation is almost impossible with an ulcer situated high up on the lesser curvature, and, on the whole, has formed an unfavourable opinion of the operation.

It is necessary to call attention to the fact that in any procedure involving resection of the lesser curvature the chief nerves are necessarily divided; and possibly certain rare sequelæ of such operations may be thus explained.



The surgical treatment of cardiospasm or idiopathic dilatation of the œsophagus is usually one of four procedures. These four are œsophago-gastrostomy, dilatation of the cardiac sphincter, œsophago-plication, and the stripping or freeing of the lower œsophagus. Dilatation is ordinarily digital, and is carried to the point of paralysis of the muscle (Walton), whilst in the other three operations a considerable denervation must be effected. Bull, in one of his œsophago-gastrostomy patients, divided the vagi, although as a rule he endeavours to avoid them; and Meyer in one case of œsophago-plication attributed the results obtained to the denervation effected, although this author later came to the conclusion that a certain proportion of his mortality in operated cases of carcinoma of the lower œsophagus was due to division of the vagi.

The operation designed by Schiassi to resect the pyloroduodenal nerves resembles in some respects that of Latarjet, in others that of Stierlin. It is probable that similar results would be obtained even if the 'circumcision' was omitted, since the section of the supraduodenal pedicle includes both parasympathetic and sympathetic nerves to the pylorus and duodenum. He has performed the operation on 26 patients with duodenal ulcer, but in 15 of these he added a gastrojejunostomy; he is satisfied that the results improve upon those obtained by routine methods. The object of the nerve resection is to give rest to the pylorus and duodenum.

Koenneke in 1922 exhaustively reviewed the surgery of the nerves of the stomach; he concluded that such operations were not justifiable. His chief objection appeared to be that, although no ill effects followed these operations on animals, yet if performed in the presence of any pathological condition this might be aggravated, and he considered that such would be the case in man. This, however, would only occur if an inappropriate operation was performed or an unsuitable case chosen, and I consider that Exner's, Bircher's, and Latarjet's results sufficiently meet this objection.

Borehers in 1921 also decided against operations upon the vagus, his reason, so far as I interpret it, being that since the vagus is not truly a motor nerve, in the same sense as a nerve to skeletal muscle is, it is unwarrantable to divide it in order to influence reflex spasm and hypermotility. I hope that in the preceding pages this criticism has been disposed of, and, moreover, that it has been shown that these operations have a wider scope than set forth by him.

Laignel-Lavastine, in a recent review of the present state of the surgery of the sympathetic system, comments favourably on the future awaiting the operative surgery of the nerves of the stomach; while Eiselsberg notes that perhaps the more radical of the present procedures may be replaced by operations upon the sympathetic nervous system of the abdomen.

### CONCLUSIONS.

A review of our knowledge of the anatomy and physiology of the nerves of the stomach has been given in the preceding pages, and an attempt has been made to correlate this knowledge with certain features of the pathology of the organ. The effects of resections of the nerves have been described

animals, and certain observations made on man have been recorded. It is evident that in animals these operations do not interfere with the general health or with the intrinsic functioning of the stomach. The results obtained by those who have operated on man support this statement. It therefore appears a logical step to investigate further the application of such surgical procedures to man.

By resection of the nerves certain effects on the stomach have been produced at will, and these may be desirable in various morbid conditions. Complete denervation results in loss of all gastric sensation and thus of referred pain, and there is also a loss of reflex control and consequently of reflex spasm; temporary diminution of motility and an increased rate of emptying occur in addition, while secretion and acidity are in all probability reduced. Vagotomy gives very similar results, but some splanchnic control remains. Splanchnotomy, by increasing vagal action, raises 'tonus' and excites the motor function as a whole. Resection of the vagal pyloric branches, or complete denervation of the pylorus, produces a semipatulous condition of the pyloric sphincter, reflex spasm is abolished, and more rapid emptying obtained. In brief, pain, motility, spasm or atony, and emptying time may be controlled to a considerable extent. The value of such changes in gastric function and sensation becomes apparent when certain morbid conditions are studied.

In considering the application of such operations, it is necessary to note that it has been shown that the nerves play a part in the development of the chronicity and complications of ulcer, if not in their actual causation. Denervation should therefore, if these premisses are correct, prove a factor in the promotion of healing and in the prevention of recurrence. A further use of these operations lies in their application for the relief of such symptom complexes as are found in the 'gastric neuroses'. If section of the nerves of the pyloric region abolishes the second phase of gastric secretion, as is believed by Lim, Ivy, and McCarthy, then denervation is definitely indicated in cases of 'hyperchlorhydria', more especially as 'hyperchlorhydria' is regarded as a precursor of gastric and duodenal ulcer. They should also be of value as supplementary procedures to a palliative gastrojejunostomy for carcinoma. The intestine can also be influenced by such nerve resections, and in the future their application may be extended to embrace derangements of the intestinal function.

Four of these operations, those of Exner, Bircher, Latarjet, and Schiassi, have already been applied to man with promising results, and their extension demands further trial. Clinical observation and the careful selection of cases should demonstrate the appropriate modification for each type of patient.

The Latarjet operation is no doubt the operation of choice when complete denervation is desired. The nerve-supply of other organs is not interfered with, and the stomach will continue to function automatically, ignoring, and ignored by, other parts. Therefore an organ, the control of which has deviated too far in either direction, will be restored to a basic condition by this operation. Resection of the vagi or splanchnics alone must be approached with more care; nevertheless, these operations may prove of great value. Subdiaphragmatic vagotomy is the simplest of those designed

to abolish vagus over-action, whilst transpleural splanchnotomy for diminished vagal control or increased sympathetic action, following the technique of Jean, is more feasible in man than any transperitoneal route.

The prophylaxis of ulcer and the cure of various of the 'gastric neuroses' are the two chief uses of these procedures; with the former they will be associated with an operation such as local excision of the ulcer. I would suggest that denervation of the pylorus, or resection of the hepatic branch of the vagus as it crosses between the layers of the gastrohepatic omentum, may, in certain instances, replace either of the operations noted above, especially for the relief of pylorospasm. It is further suggested that it is worthy of trial in the hypertrophic pyloric stenosis of infants. The operation of

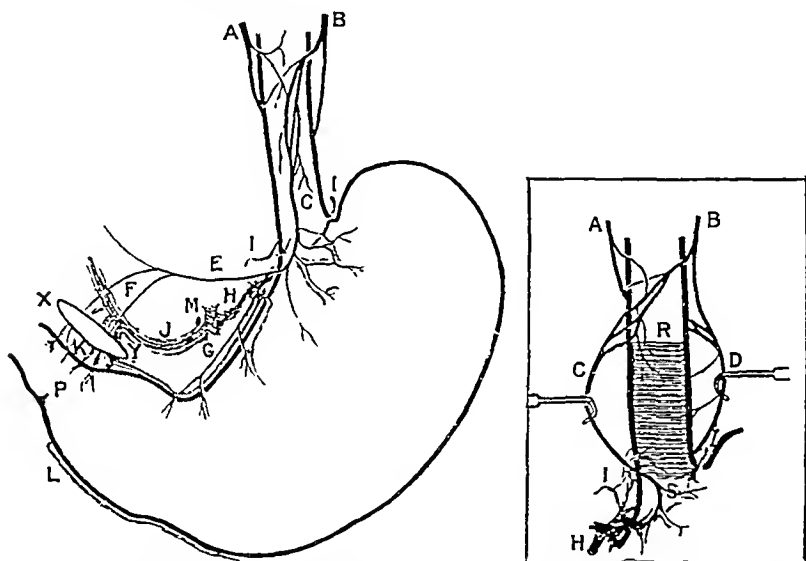


FIG. 420.—Drawing to illustrate the nerve-supply of the stomach. The site of division of the nerves in the operation of denervation of the pylorus is shown (X-Y). The inset shows the area stripped in denervation of the lower œsophagus (R-S). A, Right vagus; B, Left vagus; C, Anterior vagal trunk; D, Posterior vagal trunk; E, Hepatic vagal branch; F, Pyloric and duodenal branches; G, Principal anterior nerve of the lesser curvature; H, Sympathetic nerves with the left gastric artery; I, Sympathetic fibres with the inferior phrenic arteries; J, Sympathetic nerves with the hepatic artery; K, Pyloric sympathetic branches; L, Sympathetic fibres with the right gastro-epiploic artery; P is placed on the pylorus, and M indicates the celiac artery.

denervation consists in the division of the anterior layer of the hepato-duodenal ligament and adjoining part of the anterior layer of the gastro-hepatic omentum immediately above and parallel to the pylorus. The superior aspect of the pylorus and first stage of the duodenum are then cleared by a process of gauze and scissor dissection, either with or without ligature of the right gastric artery, and the nerve-supply is thus effectively destroyed (Fig. 420). The operation is of the shortest and simplest, and it must, *a priori*, prevent reflex pylorospasm, and produces a semipatulous condition of the sphincter. The operation recently described by Schiassi for duodenal ulcer is very similar.

Another operation which is suggested as a logical procedure is denervation of the lower œsophagus. Its application lies in the condition known as cardiospasm. So long as denervation is confined to the lower third, no ill effects should ensue. The operation may be performed either by a transpleural or transperitoneal route; of these the latter appears preferable. The lower œsophagus is freed, drawn down, and whilst the vagal trunks are displaced laterally the œsophagus is stripped in order that all nerve twigs may be effectively destroyed. Accidental wounding of the vagal trunks, should it occur, has been shown not to be of such importance as was formerly believed. The œsophagus may also be exposed by the method described by Grégoire.

Definite organic stricture is the chief contra-indication to the performance of these operations if carried out alone; pyloric stenosis is, however, the condition in which gastrojejunostomy gives its most satisfactory results. Another more theoretical contra-indication is that after denervation a serious organic lesion, such as carcinoma, may silently develop. Latarjet has recorded a case.

### SUMMARY.

Evidence has been put forward to show:—

1. That the anatomical distribution of the nerves to the stomach is sufficient to account for instances of reflex spasm or of incontinence of the pylorus in the case of gastric or duodenal ulcer, and of spasm or of atony of the body of the stomach with duodenal ulcer.

2. That the 'ulcer-bearing' area of the stomach corresponds to the area in which the nerve branches are chiefly grouped.

3. That local spasm with resultant anemia is a probable cause of the chronicity of ulcer, aided by factors such as retention and infection set up by reflex spasm.

4. That certain of the 'gastric neuroses' may be exactly simulated by the resection of either the vagi or the splanchnic nerves.

5. That the results of nerve stimulation and therefore of nerve irritation can also simulate certain of these 'neuroses'.

6. That the phenomena grouped under the title of the 'nervous dyspepsias' or 'gastric neuroses' may have at least three sources of origin; and that certain of these conditions will probably come to be regarded as pre-ulcerous. These sources are: (a) Peripheral irritation of the nerves, manifested by gastric signs and symptoms. The site of irritation may lie in the stomach and may set up both local and reflex disturbances, or be situated more remotely, as in the gall-bladder, then producing reflex gastric troubles. (b) Lesions of the nerve paths. (c) Central disorders, possibly of endocrine origin.

7. That as a logical sequence of the foregoing, operations upon the nerves are indicated in certain disorders and diseases of the stomach.

8. That such operations are feasible, that in animals their results may be forecast, and that certain of them have been performed on man.

Two operations have been suggested based on these findings; one especially suited to the condition of pylorospasm, the other to that of cardiospasm.

I desire to express my thanks to my colleagues Dr. B. A. McSwiney and Professor J. S. B. Stopford for permission to utilize in this paper the results of work performed with them. I also wish to express my indebtedness to Dr. J. M. Woodburn Morison for his assistance, especially as regards the remarks on the X-ray findings, and to Mr. G. L. Brown for his kindness in examining the stomach musculature of certain of the operated animals. For the drawings I am indebted to Professor Stopford.

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The specimen from the case which is here reported corresponded exactly to Brin's description, and seemed in general appearance to duplicate the case from Rafin's clinic illustrated in Giuliani's report,<sup>3</sup> so that it seemed advisable to report it as a 'hæmorrhagic cyst'. The subsequent investigation showed that the primary condition was an angioma, but the end-results are such as I think have never been described, and a full description may shed some light on the etiology of the excessively rare condition of 'large hæmorrhagic renal cyst'.

### CASE REPORT.

McM., age 33, labourer, was admitted to my care in the Wellington Hospital on Dec. 16, 1924. He complained that while lifting a heavy milk-can two years ago, he felt that something had given way in his side. Hæmaturia set in the same night. The blood was well mixed, and neither then nor subsequently were any clots passed. He was put to bed by his doctor, and after a few days' rest the bleeding ceased. He avoided heavy lifts for eighteen months, and then, following another 'strain', hæmaturia recommenced and has persisted almost without intermission until the time of the examination. He has never had frequency, dysuria, or any difficulty in passing water. There has been no pain in either loin. His medical attendant reports that hyaline casts were found in the urine in the interval between the first and second attacks of bleeding.

The previous history and family history present no important features. He has at times been addicted to alcoholic excess.

ON EXAMINATION.—The patient was a sturdy, well-built man, with good muscular development; well nourished, and marked absence of wasting; very anæmic. Nothing abnormal in heart or lungs, or nervous system. Muscular development prevented a very satisfactory abdominal examination, but neither kidney was palpable or tender, nor was there any tenderness or pain in the costo-vertebral angles. There was no dullness in the suprapubic region. Per rectum, the prostate, vesicles, base of the bladder, and lower end of the ureters appeared to be normal.

*Blood-pressure.*—Systolic 120, diastolic 80.

*Urine.*—Specific gravity 1018. Acid. Blood obviously present and well mixed. Albumin: a trace. Sugar: nil. Microscopically: red corpuscles, a few white, a few hyaline casts. No bacteria in smear or culture.

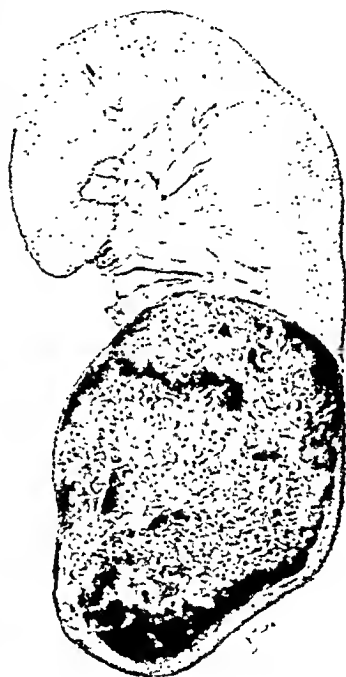
*Cystoscopic Examination.*—The urethra admitted a No. 18 F. cystoscope without difficulty. The bladder appeared to be normal in all parts, including the ureteric orifices. Efflux on the right side, every 20 seconds; on the left, every 15 seconds. Blood was easily seen in each efflux from the right side, while those from the left were clear. Indigo-carmin (10 c.c. intravenously) appeared in four minutes from the left side, in seven from the right. In the latter case the dye was in somewhat less concentration. Total kidney function: phenolsulphonephthalein, 60 per cent in two hours.

PROVISIONAL DIAGNOSIS.—'Hæmorrhagic nephritis' (essential hæmaturia) or neoplasm. The presence of casts, the long duration, and the complete absence of clots favoured the former.

The patient was so exsanguinated that further delay for investigation seemed dangerous, and operation was advised.

**OPERATION.**—Under nitrous oxide and ether the kidney was exposed by the method of Bazy, the peritoneum at the inner angle of the wound being opened in order to ascertain the absence of metastases in glands and other organs. The opening in the peritoneum was then closed, and the right kidney removed extraperitoneally. The lower pole, which was cleared with some difficulty, was hard, white, and covered with dense fibrous tissue. It had one or two small bosses upon it. Owing to the thick wall and the pressure of the contained fluid, the impression was given that one was dealing with a solid tumour. The mass extended to a little above the hilum, and above this level the kidney appeared to be normal. Access to the pedicle was somewhat difficult, as the hilum lay in a deep recess formed by the angle between the large mass below and the upper pole above, while the renal vein was very short. An accessory artery going to the upper pole caused some embarrassment before hæmostasis was secured. A pint of whole blood was given intravenously and the patient left the table in fair condition. Recovery was rapid and convalescence uneventful.

**The Specimen.**—The upper pole is normal in size and appearance, and the fibrous capsule thin and smooth. The lower pole (*Fig. 421*) is entirely occupied by a cystic tumour 11 cm. in diameter. The wall of the cyst is definitely fibrous, and varies in thickness between 1 mm. and 2.5 mm. On incising the tumour, 250 c.c. of dark fluid blood and tarry clot escaped. The internal lining of the cyst is smooth and glistening, except where masses of fibrin and clot are adherent. No dilated veins and no focus of bleeding are apparent in the cyst wall. The upper major calix of the kidney pelvis is normal, the lower is absent. A slight connection exists between the upper part of the cyst and the pelvis through which a bristle can be passed. At two points bosses project externally. At the upper part the cyst is separated from the rest of the kidney substance by its laminated fibrous wall, and the part of the kidney immediately adjacent to this appears to be pale and compressed. The internal measurements of the cyst are 9.2 cm. from above down, and 6.8 cm. in the other two diameters. The whole was covered by the fibrous capsule of the kidney, which passed without interruption into the capsule of the sound part of the organ.



*Fig. 421.*—Hæmorrhagic cyst in lower pole of right kidney.

**Microscopical Examination.**—The cyst wall shows much free and partly organized blood, beneath which are a series of large intercommunicating blood spaces lined by endothelium (*Fig. 422*). In those parts which appear

to have been subjected to pressure, the blood spaces are obliterated, and there are then seen close masses of spheroidal cells with fine slit-like spaces throughout. Lying in the midst of and at the periphery of the cyst lining there is much old blood pigment, some of which lies in phagocytes. In no place is there any infiltration of the capsule, which consists of laminated fibrous tissue. Near the junction of the kidney proper and the tumour there can be seen flattened and compressed glomeruli in the fibrous capsule. The adjacent kidney cortex shows infiltration with small groups of inflammatory cells."

In certain places what appeared to be the original lining of the lower calix in the shape of definite transitional epithelium could be seen, outside the angiomatous structure. Outside the laminated fibrous wall of the cyst definite compressed renal tissue could be made out, and, beyond that again, the fibrous capsule of the kidney.

The specimen was reported as a *cavernous hæmangioma* giving rise to

a blood cyst. It is probable that an angioma arising in one of the papillæ had bled into the lower major calix; then the outlet of the latter into the main pelvis became obliterated either by the growth or by organized clot. In consequence distention gradually took place, and this, either by itself or aided by the progress of the tumour, obliterated the minor calices, and the reaction of the tissues by reinforcing the wall of the large calix formed the laminated fibrous cyst wall as found. This sequence of events seems to be a reasonable supposition.

The description of the microscopical examination is that of Dr. P. P. Lynch, Pathologist to the Wellington Hospital, to whom

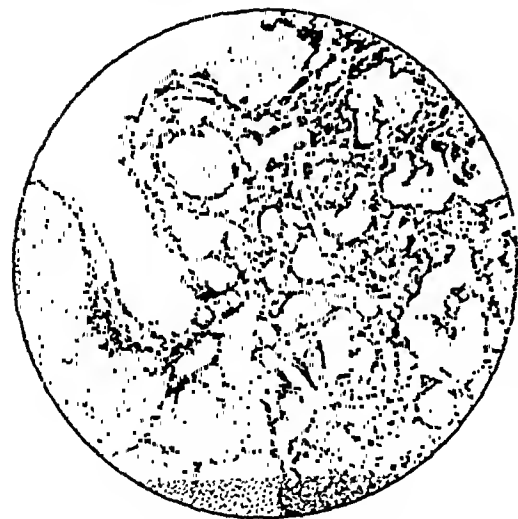


FIG. 422.—Section through angioma in wall of cyst, showing wide blood spaces.

I am indebted for a careful examination of the slides and specimen.

The analogy between this case and that of Giuliani mentioned above is striking, not only in the general similarity in appearance of the specimens, but also in the fact that both were accompanied by hæmaturia. In fact, Giuliani's case was the only one in Brin's series that presented this symptom. To the naked eye both specimens, to judge from the illustration, are identical, and in the case of McM. it was only by a somewhat searching microscopical examination that the angiomatous formation was found. It is possible to conceive that, with the growth of the cyst, cellular elements that might have been concerned in its origin could disappear, so that eventually there would be no evidence that the cyst arose from an angioma.



I think that these two cases must be considered as examples of partial hæmatonephroses, due to bleeding from an angioma into the lower major calix, with results outlined above. The condition of partial hydronephrosis is rare, but a case is reported by Bazy.<sup>4</sup> It seems to me that the closing of the outlet of one department of the pelvis would more probably take place where some neoplastic or clot formation was present than in a simple case, but either condition must be of excessive rarity.

**Etiology of Hæmorrhagic Cysts.**—Most of the theories of origin hitherto suggested have been unsatisfactory and inapplicable. The following suggest themselves for critical examination :—

1. *Bleeding into a Solitary Serous Cyst.*—It has been frequently stated that hæmorrhagic cysts are merely a form of serous cyst where bleeding has occurred. While it is no doubt true that the fluid in some of these cysts is blood-stained, their nature is totally different. The presence of the thick fibrous wall, compressed renal substance, and the fibrous capsule of the kidney itself, between the cyst content and the kidney surface—these are features quite foreign to the simple cyst.

2. *Trauma Causing an Effusion of Blood into the Kidney Substance with Encapsulation.*—The history of trauma is absent in most of the cases, and, even where it occurs, the bleeding would cease, and the resulting cyst contain the usual straw-coloured fluid common to old traumatic blood effusions, while a history of hæmaturia at some time would be the rule. Continuity of bleeding, and not only a single hæmorrhage, is essential for the formation of the large blood cysts.

3. *Aneurysm of one of the Minor Renal Vessels.*—The appearance of the cyst wall with its smooth surface and clinging masses of fibrin might suggest the possibility of this explanation; but apart from the difficulty in conceiving that such a tiny vessel should expand to this size, pulsation has never been recorded, nor is it likely that an aneurysm would limit itself so exactly to the confines of the kidney.

4. *Partial Hæmatonephrosis.*—By far the most probable explanation to my mind of all the cases is the one suggested previously, namely, partial hæmatonephrosis. The bleeding may have taken place as the result of an angioma as in my case, or followed one of the various obscure local conditions that, apart from 'nephritis', may cause 'essential hæmaturia'—varices round a papilla, minute papillomata, etc.

In the vast majority of cases the blood would pass into the pelvis without retention; but in some few either the factor that causes partial hydronephrosis may be present—whatever that may be—or the narrow outlet may be closed with clot adhering to the pelvis lining. In this case the gradual organization and augmentation of the clot would cut off the affected calix—major or minor from the main pelvis, and the continued bleeding would cause distention of the closed cavity. Slight hæmaturia which would be present in the initial stages might easily pass unnoticed.

Apart from this distention of an excluded calix, it is quite conceivable that a small angioma might bleed into the kidney substance, causing a cyst, and that the neoplasm, while still present about the wall of the cyst, might be obscured in masses of organized clot and fibrin. The furious bleeding that

occurred in some of the cases where an attempt was made to dislodge these fibrinous masses is confirmatory of the hypothesis.

The etiology suggested would readily account for the layer of compressed renal substance that surrounds the cysts. In some cases of partial hæmatonephrosis a severe muscular strain, causing compression of the enlarged kidney, might re-open a passage to the pelvis, and bleeding would continue to take place externally, producing hæmaturia. This seems to be the only explanation in the case of McM.

**Diagnosis.**—This is similar to that of serous cysts except where hæmaturia is present. In the latter case the bleeding kidney is located and the condition discovered at operation. In the absence of palpable tumour or hæmaturia, more relative decrease in function may be expected than in the case of serous cysts, as more of the kidney substance is involved. Pyelography is more likely to give information, as some portion of the renal pelvis is almost certainly involved.

Conditions to be distinguished from 'large solitary hæmorrhagic cysts' are :—

1. *Cystic Degeneration of a Neoplasm, usually a so-called Hypernephroma.*—Here the lining is ragged and composed of tumour substance, while the presence of a growth is as a rule obvious. There is a complete absence of the very definite fibrous lining, smooth and even glistening on its inner surface, in parts where clot is absent, which is one of the characteristics of blood cysts.

2. *Hæmatonephroses.*—A number of cases where a hydronephrosis has contained bloody fluid have been encountered, and this is not surprising when one considers the frequency with which bleeding is associated with simple hydronephrosis, and that not only when the latter empties itself, but even when it is of the closed type. There would be no difficulty in differentiating such a condition involving the whole kidney from a localized cyst.

3. *Traumatic Pararenal Cysts.*—These are not covered by the fibrous capsule of the kidney in continuity as in the case of the true cyst.

**Treatment.**—If the cyst has attained any size, operation will clearly be indicated, either on account of pressure symptoms or to clear up the diagnosis. In the case of hæmaturia interference for the latter reason is imperative, and in some cases will be urgent, in order to stop hæmorrhage.

The conservative operations so frequently attended with brilliant results in the case of simple cysts have no place here. In favourable cases partial nephrectomy might conceivably be sufficient if the real condition were known; but it is just this and the etiology that must be so much in doubt, that only complete nephrectomy will satisfy the conscience of the surgeon. Where attempts have been made to turn out the cyst contents, alarming hæmorrhage has always necessitated a rapid nephrectomy—a fact which points to an angiomatous basis for the condition.

## SUMMARY.

1. Large solitary hæmorrhagic cysts (*les grands kystes hématiques*) are excessively rare. They have few characteristics in common with solitary simple cysts (*les grands kystes séreux*), and should be placed in a separate category.

2. They are different in nature from blood cysts caused by the degeneration of malignant tumours, from hæmatonephroses, and from traumatic pararenal cysts.

3. The suggestion is made that these cysts are of the nature of partial hæmatonephroses, where the source of the bleeding remains active over a long period of time. This source may be in the papillæ of the involved portion of the pelvis, and in the case reported an angioma was demonstrated.

4. In some instances the cyst may owe its origin to an angioma in the kidney substance itself.

5. The diagnosis is obscure, and the treatment is total nephrectomy.

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<sup>1</sup> BRIN, *Encycl. franç. d'Urologie*, iii, 19.

<sup>2</sup> SOULIGOTX and GOUTET, "Contribution à l'Étude des grands Kystes hématiques simples du Rein", *Arch. gén. de Méd.*, 1882, ii, No. 14, 833.

<sup>3</sup> GIULIANI, *Jour. d'Urologie*, 1913, March, 175.

<sup>4</sup> BAZZ, *Encycl. franç. d'Urologie*, iii, 175.

## OBSERVATIONS BEARING UPON THE OPERATION OF PROSTATECTOMY.\*

BY I. COHEN, E. C. DODDS, and C. H. SHORNEY WEBB, LONDON.

THE majority of surgeons will admit the difficulty of ascertaining the state of the renal function from a purely clinical inquiry, and they will agree that some additional test in the form of a laboratory examination is necessary. Owing to the variety of tests at present in use it is very difficult for any single observer to possess an intimate knowledge of all, with the result that each worker adheres to some test of which he has special experience. It will perhaps be as well to review very briefly these various tests, and to epitomize the current opinion upon them. It is not proposed here to enter into a detailed classification of the various tests, but merely to refer to them singly.

To commence with, practically all authorities are agreed that the mere examination of the urine for albumin, casts, pus, organisms, and other abnormal constituents, although important, is not reliable as a guide to operative prognosis, since it is quite possible to have very advanced renal changes coexisting with slight urinary alterations. Moreover, the results of back-pressure from prostatic enlargement or other causes rarely produce any marked changes in the urine.

A series of very important tests depends upon the administration of some natural or foreign substance and noting the excretion of the body in question. These are of two main varieties: first, those in which dyes are given, and, secondly, those employing substances such as urea, benzoic acid, phloridzin, etc. The dye tests in general use are the indigo-carminic and the phenolsulphonephthalein reactions. The former is usually performed clinically, and the excretion of each kidney can be investigated, either by means of the ureteric catheter or the cystoscope.

That the performance of some such test as this is an absolute necessity when dealing with unilateral lesions no one will doubt. In fact, this type of method constitutes the only one by which the individual functions of the kidney can be judged. The phenolsulphonephthalein test has proved reliable, but suffers from certain serious drawbacks. Thus the presence of blood in the urine renders the colorimetric estimation of the dye content exceedingly difficult, if not impossible.

Another test, used extensively in England, is the urea-concentration test of H. MacLean.<sup>1</sup> This test is of proved value in medical cases, but its value, when taken alone, in cases with obstruction, is not quite so certain. In *Table I* can be seen a comparison between the urea-concentration test and

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\* From the Biochemical Department of the Middlesex Hospital, London.

the figures obtained by blood analysis. By means of the administration of 15 gm. of urea, and the collection of the urine in three subsequent hourly specimens, the functional capacity of the kidney can be judged from the percentage of urea in the specimen.

*Table I.*—BLOOD-UREA AND NON-PROTEIN NITROGEN AS COMPARED WITH THE UREA-CONCENTRATION TEST.

CASE	BLOOD-UREA	BLOOD N.P.N.	URINE-UREA CONCENTRATION PER CENT		
			Before Urea	1 Hour after	2 Hours after
	Mgram. per 100 c.c.	Mgram. per 100 c.c.			
C. P.	32	63	2.6	3.2	3.5
T. L.	25	67	3.1	3.5	3.5
S. C.	40	64	0.9	1.2	1.3
A. H.	33	60	1.8	2.0	2.5
C. E.	70	66	4.2	3.8	4.4
J. W.	68	71	1.3	1.7	1.8
F. B.	28	42	1.1	1.4	1.9

To summarize briefly, it would appear that the mere examination of the urine alone does not form a sufficiently accurate means of estimating the renal function in relation to surgical conditions.

A whole series of new tests was opened up by the introduction of relatively simple and accurate methods of blood analysis. Mainly owing to the work of Otto Folin<sup>2</sup> and his school, it has been made possible to analyse about 10 c.c. of blood for a whole series of important constituents. Whereas the methods mentioned so far have tested the kidney as an eliminatory organ, any blood observation will measure retention of certain constituents due to some defect in kidney function. The blood content of the following substances has received most attention. They are given below, together with the normal percentage in the blood:—

Urea .. ..	20 to 40	mgram. per 100 c.c. of blood
Non-protein nitrogen .. ..	20 to 40	" " "
Creatinine .. ..	1 to 1.5	" " "
Uric acid .. ..	2 to 3	" " "
Amino-acid nitrogen .. ..	4 to 7	" " "
Sugar .. ..	80 to 120	" " "
Chlorides as NaCl .. ..	400 to 500	" " "

The estimation of each of these substances has been claimed as the ideal kidney function test by different authorities. Thus MacLean (*loc. cit.*) and De Wesselow,<sup>3</sup> in their books, review the literature very fully, and maintain that the urea content of the blood is the most valuable guide to prognosis. The latter authority, in considering the advisability of an operation for

removal of the prostate, states: "In cases in which the blood-urea content exceeds 50 mgrm. per 100 c.c., a two-stage operation is advisable"; and if the figure lies between 35 and 50 mgrm. per 100 c.c., clinical evidence and other tests must be relied upon.

The American workers maintain that a more complete examination of the blood should be performed. Folin (*loc. cit.*) suggests that all the constituents tabulated above should be estimated, and opinion should be passed upon the general consideration of the nitrogen distribution.

Frontz and Geraghty<sup>4</sup> state that the most useful information is gained from the non-protein nitrogen and uric-acid contents. They disregard urea. Myers and Finc,<sup>5</sup> and Berglund<sup>6</sup> suggest that the estimation of creatinine is of the greatest importance, and that a figure of persistently over 1.5 mgrm. per 100 c.c. must be regarded as evidence of serious renal damage.

It is necessary to distinguish between nitrogen retention due to permanent destruction of kidney substance as seen in chronic nephritis, and that due to back-pressure from some genito-urinary obstruction. In the latter case continued obstruction eventually leads to permanent impairment of function, but the removal of the obstruction to urinary outflow will, if performed early enough, lead to an improvement in the excretory power of the kidney. Johnson's<sup>7</sup> experiments showed that after tying the ureter, and thus causing complete obstruction, function could be regained even after fourteen days when the ligature was removed.

Many of our cases as recorded later showed marked decrease in nitrogen retention where bladder drainage was resorted to. It is conceivable that tests like that of MacLean might be obscured by the difficulty in efficient emptying of a vesiculated bladder. Coming to the blood estimation, the argument that the figures obtained may give no information in unilateral lesions does not hold when the question is only whether there is enough acting kidney substance in the body to warrant the risk of the operation of prostatectomy.

The investigations about to be described were commenced three years ago. It was felt that there was insufficient knowledge on the problem to rely on any one blood constituent, and that the best interests would be served by performing as complete analyses as possible in every case. By this means it was thought that after a large number of cases had been examined the diagnostic significance of each constituent would be judged. Where pre-operative figures were raised, wherever possible we suggested drainage of the bladder by means of a suprapubic cystotomy before proceeding with prostatectomy.

Out of our series of 69 cases, in 54 all the blood constituents mentioned earlier were estimated. In the others only urea, or urea and non-protein nitrogen determinations, could be made, owing to small quantities of blood. It was decided to classify the results into three groups: (1) Those in which only one blood analysis was performed; (2) Those showing no benefit from suprapubic drainage; (3) Those in which the effect of suprapubic drainage was found to be successful, as judged by two or more analyses. Owing to the large number of figures, it was found possible only to publish representative examples of each group.

*Group I.*—With regard to this group—that is, those cases in which only one blood analysis was performed—*Table II* shows the result of blood analyses upon a series of unselected cases prior to operation. For one reason or another a second blood analysis could not be performed. It will be noted that in some cases prostatectomy was performed, although the blood figures were in the danger zone. In other cases drainage, either temporary or permanent, was instituted, although the figures were well within

*Table II.*—CASES WITH ONE SET OF FIGURES.

CASE	UREA	N.P.N.	CREATININE	URIC ACID	AMINO-ACID N.	SUGAR	CHLORIDES	REMARKS
	Mgmm. per 100 c.c.	Mgmm. per 100 c.c.	Mgmm. per 100 c.c.	Mgmm. per 100 c.c.	Mgmm. per 100 c.c.	Mgmm. per 100 c.c.	Mgmm. per 100 c.c.	
W. F.	34.0	37.0	2.0	2.5	5.0	125	462	Permanent drainage
C. P.	32.0	63.0	1.6	3.7	11.0	99	462	Prostatectomy
W. P.	23.0	67.0	1.5	2.7	7.0	134	450	Prostatectomy
T. L.	25.0	67.0	1.6	2.9	11.3	143	379	Prostatectomy
G. G.	32.0	29.0	1.4	1.6	5.4	112	388	Patient unsuitable for prostatectomy. Double Steinach operation performed
A. H.	33.0	60.0	—	—	—	114	—	Suprapubic cystotomy and prostatectomy 7 days later
W. H.	52.0	53.0	1.1	2.8	7.6	98	413	Cystotomy and prostatectomy 16 days later
F. H.	41.0	55.0	1.9	2.6	10.0	103	429	Suprapubic prostatectomy
C. Q.	29.0	40.0	1.6	4.0	5.9	101	462	Expectant treatment. Patient unsuitable for operation
C. K.	38.0	48.0	2.0	2.6	8.1	87	470	Suprapubic cystotomy
E. B.	30.0	53.0	1.7	2.6	8.2	98	—	Prostatectomy
J. M.	97.0	82.0	2.3	4.9	5.9	146	445	Cystotomy. Died of uremia

normal limits. In these instances the surgeon was guided by the general condition of the patient irrespective of laboratory findings. One of the first points striking the observer is that the values for the various constituents do not rise or fall together. Thus, in one case the blood-urea and non-protein nitrogen contents may be low, whilst the creatinine is high, or vice versa. The sugar content varies quite independently of the renal condition, whilst the chlorides and amino-acid nitrogen bear little or no relation to the clinical condition.

It would appear, therefore, that only the urea, non-protein nitrogen, creatinine, and uric acid remain for consideration. If the table be inspected further, it can be seen that the non-protein nitrogen and uric acid content vary in roughly the same manner, and that the creatinine content is always above normal when the former are high, but the reverse does not necessarily hold true. Although in advanced conditions the urea content is high, many of the cases show a normal content, even when the other constituents are very much above normal. This would narrow the choice down to non-protein nitrogen and uric acid. A careful inspection of all the figures will show that these two values are the most constantly raised in conditions associated with renal disturbance, and, if these be taken as a guide, it might reasonably be expected that as few mistakes as possible would then be made.

After having decided that the non-protein nitrogen and uric acid content are the most reliable, the next problem is to fix danger limits for operation.

*Table III.*—CASES NOT YIELDING

CASE	BEFORE CYSTOTOMY						
	UREA	NON-PROTEIN NITROGEN	CREATININE	URIC ACID	AMINO-ACID NITROGEN	SUGAR	CHLO.
R. B.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. 100
	38.0	50.0	1.1	3.1	6.6	120	28
	6 days later						
	64.0	50.0	1.2	3.3	—	87	48
F. B.	28.0	42.0	1.1	4.1	6.8	86	49
G. N.	60.0	77.0	1.9	3.1	6.4	85	5
J. L.	119.0	118.0	2.1	5.9	4.4	127	4
W. M.	No figures before drainage						



*Group II.*—In *Table III* are included the results from a series of cases either proving fatal or not yielding to suprapubic drainage.

*Case W. M. (Table III)* shows a series of analyses upon a man after suprapubic drainage. After ninety days the non-protein nitrogen was much above normal (76 mgrm. per 100 c.c.), whilst the urea was normal. He was operated upon, a suprapubic prostatectomy being performed, and he died immediately after.

At the post-mortem examination the kidneys showed advanced fibrotic changes.

*Case G. N. (Table III)* showed a fall in his urea content from 60 to 51 mgrm. per 100 c.c., but the non-protein nitrogen remained 60 mgrm. per 100 c.c. Prostatectomy was performed, and he died of uræmia nine days later.

We have examined a series of nine fatal cases, in all of which the non-protein nitrogen content was above 60 mgrm. per 100 c.c.

## SUPRAPUBIC DRAINAGE.

AFTER CYSTOTOMY							REMARKS
UREA	NON-PROTEIN NITROGEN	CREATININE	URIC ACID	AMINO-ACID NITROGEN	SUGAR	CHLORIDES	
Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	
—	—	—	—	—	—	—	—
52.2 37.8	54.6 32.2	— —	— —	— —	— —	— —	Suprapubic prostatectomy
25.8	42.3	1.5	3.9	6.9	115	379	Cystotomy performed, prostatectomy being difficult
61.2 51.4	61.5 60.3	— 2.1	— 2.8	5.0 7.2	110 100	— —	Died after prostatectomy done nine days after last figure
131.9	137.5	2.9	4.5	8.4	105	429	Died of uræmia
159.3 110.7 90.0 112.0 114.0 84.0 36.0	106.7 85.3 87.5 84.0 80.0 66.3 75.6	— — — — — — —	— — — — — — —	— — — — — — —	— — — — — — —	— — — — — — —	Treated as in-patient
							Treated as out-patient Admitted and operated on Died of uræmia

*Group III.*—On turning to *Table IV*, which deals with cases operated upon successfully and yielding to suprapubic prostatectomy, it can be seen that suprapubic drainage produced a marked reduction in the figures, and

Table IV.—CASES YIELDING

CASE	BEFORE CYSTOTOMY					
	UREA	NON-PROTEIN NITROGEN	CREATININE	URIC ACID	AMINO-ACID NITROGEN	SUGAR
	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.
J. S.	44.0	51.0	1.3	2.8	6.3	114
A. W.	161.0	141.0	1.1	2.5	11.2	143
H. S.	36.0	58.0	1.3	2.6	5.1	113
F. S.	29.0	81.0	—	—	—	—
L. W.	49.0	42.0	1.3	2.6	5.3	98
A. S.	68.0	133.0	—	—	—	—
C. E.	70.0	66.0	1.6	2.3	6.3	106
F. C.	71.0	77.0	—	—	—	—
F. W.	34.0	52.0	1.3	2.3	4.8	106
R. G.	121.0	109.0	3.1	3.8	7.9	133

### DISCUSSION OF RESULTS.

An examination of the results points to the fact that the retention of non-protein nitrogen and uric acid is far more constant in cases of renal damage (as judged by post-mortem and clinical post-operative evidence) than the other constituents. By far the most important object is to fix a definite figure at which a two-stage operation should be advised. It would appear that the danger point lay round 50 mgrm. per 100 c.c. for non-protein nitrogen, and about 3.5 mgrm. per 100 c.c. for uric acid content. It must be admitted that occasional odd cases have gone through perfectly normally with figures above these, but against these must be contrasted the number who have either died or exhibited serious clinical symptoms.

As the result of these investigations we have worked upon the rule that figures of above 60 mgrm. and 3.5 mgrm. per 100 c.c. of non-protein nitrogen and uric acid respectively, indicate that a suprapubic cystotomy should be

that before the final operation for enucleation, the non-protein nitrogen content was below 60 mgrm. per 100 c.c.

## SUPRAPUBIC DRAINAGE.

AFTER CYSTOTOMY							
PER- IOD	UREA	NON-PROTEIN NITROGEN	CREATININE	URIC ACID	AMINO-ACID NITROGEN	SUGAR	CHLORIDES
	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.	Mgrm. per 100 c.c.
1st	23.0	41.0	1.1	2.3	5.9	9.8	339
2nd	29.0	40.0	1.2	2.8	—	116	519
3rd	35.0	30.0	1.3	2.1	6.0	114	476
4th	31.0	32.0	—	—	—	—	—
5th	32.0	33.0	1.3	2.3	5.8	122	495
6th	38.0	48.0	—	3.4	6.8	—	—
7th	44.0	61.0	2.1	2.8	6.9	103	479
8th	48.0	69.0	—	3.3	8.5	97	—
9th	30.0	50.0	—	—	8.0	98	—
10th	38.0	45.0	—	—	—	141	—
11th	42.0	49.0	1.5	1.8	8.5	96	363
12th	33.0	44.0	—	—	—	—	—
13th	24.0	38.0	2.4	3.6	5.4	79	485
14th	61.0	59.0	2.0	3.2	4.9	98.5	528

performed, and, after drainage, the blood figures will usually sink to below these limits, when enucleation may be performed. When the high figures persist in spite of this preliminary treatment, any major operation is contra-indicated, as the kidneys have become permanently damaged.

That the striking clinical benefit so frequently seen after a suprapubic cystotomy is accompanied by a corresponding improvement in the nitrogen retention can easily be seen from *Table IV*. In one case, A. W. (*Table IV*), the figures before the operation were 161 mgrm. of urea and 140 mgrm. non-protein nitrogen respectively. Fourteen days after operation these had fallen to 29 and 39 mgrm. per 100 c.c. respectively. Cases of this nature are extremely common, and in the majority of patients suprapubic drainage is followed by almost immediate improvement.

It would appear that the actual operation for prostatic enucleation adds about 20 mgrm. to the figures for urea and non-protein nitrogen; if then they were high before, the increase may be sufficient to endanger the patient.

With regard to the relative advantage of purely clinical observations over purely laboratory tests, it is a difficult matter for us to express a definite opinion.

*Table V.*—COMPARISON OF BLOOD-UREA AND NON-PROTEIN NITROGEN FIGURES WITH DURATION OF SYMPTOMS.

CASE	AGE OF PATIENT	DURATION OF SYMPTOMS	BLOOD	
			Urea	Non-protein Nitrogen
			Mgrm. per 100 c.c.	Mgrm. per 100 c.c.
W. F.	72	2 months	34.0	37.0
A. G.	51	10 months	39.0	86.0
J. W.	71	12 months	80.0	113.0
R. B.	71	2 years	38.0	50.0
W. R.	78	5 years	102.0	—
W. H.	67	6 years	52.0	58.0
F. B.	57	7 years	28.0	42.0

A study of the history of our cases would seem to indicate that the duration of the complaint plays a very small part in determining the amount of retention, as will be seen in a few cases shown in *Table V*. It is also remarkable that these back-pressure cases often show slight general symptoms with urea and nitrogen figures as high as those usually associated with uræmia, for example:—

*Case A. W. (Table IV):* Urea 161, non-protein nitrogen 140.

*Case R. G. (Table IV):* Urea 121, non-protein nitrogen 109.

There can of course, be no doubt but that the closest co-operation between the laboratory and the surgeon is the only method of reducing errors of judgement in these cases to a minimum.

### CLINICAL ASPECT.

The operation of prostatectomy may be productive of considerable shock to the patient—shock of two kinds: (1) General surgical shock such as may be produced by almost any major operation; (2) Urinary shock such as may be directly consequent upon any major operation on the urinary tract, ending possibly in uræmia. Clinicians often lose sight of the importance of the first type, in their eagerness to investigate the possible occurrence of the second.

It is without doubt true that a knowledge of the state and efficiency of the kidneys is of paramount importance in determining whether the removal

of the prostate is likely to induce much or little urinary shock—with possibly complete or partial suppression of urine. It is also wise to bear in mind the injurious effect of a general anæsthetic upon an inefficient kidney. An estimation of the 'renal efficiency' is always necessary in order that the surgeon may decide whether the removal of the prostate is feasible, and whether the removal should be performed in one or two stages.

It is not, however, altogether enough to rely on the laboratory figures of the renal function in order to arrive at a decision as to whether the prostate can be removed or as to how it should be removed. The clinical picture of the patient is of as great an importance in arriving at an accurate estimation of his chances of survival after prostatectomy as in any major operation unconnected with the urinary tract. The clinical signs and symptoms which are of value in this estimation of the suitability of a case for a one- or two-stage operation are best grouped as given in the list below. Many of these only make their appearance when there is a fairly high degree of renal inefficiency present. Absence of such signs or symptoms favours a good prognosis, presence of them engenders caution.

1. *Age*.—This, being comparatively obvious, need not be enlarged upon.

2. *The Prostate*.—Some enlargements of the prostate lend themselves more easily to removal than others. Smooth, movable, elastic, moderate-sized enlargements are removed with greater facility than the small harder varieties, for instance. A large gland often shells out more easily than a small one.

3. *Desiccation*.—The patient is sometimes a dried-up shrivelled old man, with a dry wrinkled skin and little or no subcutaneous fat. Complaining of thirst, he shows a dry brown tongue and possibly cracked lips. The urine in this type of case is often of low specific gravity.

4. *Nutrition*.—Complaining of anorexia, nausea, and sometimes vomiting, the patient may be badly nourished and lacking in stamina, feeble and tottery. Constipation may be very troublesome.

5. *Fatigue*.—Sleeplessness by night, due either to frequent micturition or a definite insomnia, and drowsiness by day are not uncommon. There may be a low form of muttering delirium at night, with perhaps picking at the bedclothes. These mental symptoms may be intensified by the worry of an impending operation. Tremors of the hands and fingers may be present.

6. *Infection*.—Cystitis, pyelitis, *B. coli* infection of the urinary tract, calculi, prostatic or vesical, complicating prostatic enlargement, will generally influence the surgeon in the direction of a two-stage operation.

7. *Heart, Blood-vessels, Lungs*.—Cardiac disease, arteriosclerosis, and chronic pulmonary conditions must all be considered.

8. *Temperament*.—Best summed up in two words—fear and phlegm. The phlegmatic individual makes a far better patient than the nervous, timid type.

Laboratory investigations into the amounts of non-protein nitrogen, of blood-urea, or of uric acid will give no information as to the temperament of the patient, the state of his heart, blood-vessels, or lungs, his physical nutritional state or stamina, his robustness or feebleness, or, even less, the

condition of his prostate. The true perspective is to be found, first, in realizing the importance of the laboratory findings and in visualizing them in terms of renal function only, and, secondly, in turning elsewhere—i.e., to the bedside of the patient—for the absence or presence of those signs and symptoms upon which the most accurate estimation of the patient's chances of recovery from the two distinct forms of post-operative shock may be based. No wise surgeon allows the contemplation of his patient's kidneys to mask the picture of his patient as an individual.

### SUMMARY.

1. Owing to the lack of uniformity of views upon the value of renal function tests in relation to prostatectomy, an investigation of a series of cases was instituted.

2. For various reasons detailed in the text it was decided to concentrate the observations upon blood analysis.

3. From a study of the figures obtained in relation to after-history, it was found that the non-protein nitrogen content and uric-acid content of the blood formed the best guide to prognosis.

4. The upper limit of safety for the complete operation of prostatectomy was found to be 50 mgrm. per 100 c.c. for non-protein nitrogen, and 3.5 mgrm. per 100 c.c. for uric acid.

5. Experience has shown that if figures above these are encountered, the performance of a preliminary suprapubic cystotomy is advisable. The enucleation can then be carried out when the figures fall within the limits of safety.

6. The blood-urea content is much more unreliable than the non-protein nitrogen and uric-acid contents. A high blood-urea content must always be regarded as a serious sign, but reference to the tables will show that a low urea content cannot always be regarded as an indication of a normal kidney function.

7. Laboratory findings should never be relied upon alone, but should always be considered in combination with the clinical state of the patient.

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## PRIMARY TUMOURS OF THE URETER.\*

By R. LESLIE STEWART, EDINBURGH.

PRIMARY tumours of the ureter are of rare occurrence. Writing in 1922, Aschner<sup>1</sup> collected the comparatively small number of forty-seven published cases, illustrative of the various types of epithelial tumour which have been observed. In the available literature the present writer has been able to find the records of a further five cases. From the clinical standpoint such tumours are of considerable surgical interest on account of the grave symptoms they may produce, the difficulties in accurate pre-operative or even operative diagnosis, the frequency of malignant change, and, lastly, the radical operative measures usually required in their treatment. For these reasons the following case appears worthy of record, illustrating as it does many features in the symptomatology, diagnosis, and pathology characteristic of the condition.

## REPORT OF CASE.

A married woman, 75 years of age, was admitted to the Edinburgh Royal Infirmary on Nov. 22, 1924, being referred to the wards under the charge of Professor D. P. D. Wilkie. Her complaint was pain on the right side and hæmaturia, commencing eight months previously.

**HISTORY.**—For many years she had been subject to bronchitis and asthma. At intervals for about twenty years she had experienced attacks of hypogastric pain associated with dysuria and frequency of micturition. These attacks were generally very transient, usually nocturnal, and appeared to be brought on by cold. There had been occasional lack of vesical control, especially related to fits of coughing. A uterine prolapse complained of some years ago was relieved after wearing a pessary for a few months. During the last two years there had been chronic dyspeptic symptoms, associated with flatulence and constipation.

**PRESENT ILLNESS.**—In March, 1924, she was feeling somewhat run down in health. One morning she was attacked with sensations of shivering, malaise, and general pains, which appeared at first to be heralding the onset of influenza. Later in the day a pain commenced in the right loin, gradually increased in severity, and was felt shooting down towards the groin. Hæmaturia accompanied these symptoms, the urine being deeply blood-stained, but apparently no clots were passed. Varying in intensity, the pain and hæmaturia continued for three weeks, and were associated with frequency of micturition, especially if the pain was severe. In June there was another, but less severe, attack of hæmaturia and pain, which lasted for a week. There was no further obvious bleeding, but she complained of intermittent right-sided pain, which became severe in November. The urine was generally clear, but at times was noted to be turbid. Intermittent frequency of micturition was present, but the total amount of urine was not increased. She now noticed some slight loss of weight.

**ON ADMISSION.**—The patient was found to be a rather feeble old woman, bronchitic, with a poor appetite, and complaining of flatulent dyspepsia. The pulse was regular, with a well-sustained wave, and the arterial walls, as estimated from

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\* From the Department of Surgery, University of Edinburgh, and the X-ray Diagnostic Theatre, Royal Infirmary, Edinburgh.

the radial at the wrist, were slightly thickened. Systolic blood-pressure was 140 mm. Hg. Abdominal examination revealed no renal enlargement. There was no superficial or deep hyperæsthesia, but tenderness was present on deep palpation in the right hypochondriac and lumbar regions. The liver was not enlarged, and there was no ascites. Splashing could be elicited in a somewhat dilated stomach. Chemical examination of the urine, beyond demonstrating a very faint trace of albumin, revealed no other abnormal constituent. The Wassermann reaction was negative.

**CYSTOSCOPIC EXAMINATION.**—On Nov. 24 a cystoscopic examination was carried out. The urine withdrawn from the bladder by a catheter appeared healthy, and was retained for purposes of examination. The bladder was then filled, and it



FIG. 423.—Prior to operation. Radiogram with opaque ureteral catheters introduced. The catheter on the right side is arrested at the level of the brim of the true pelvis.

was found to hold 300 c.c. without discomfort. An examining and catheterizing cystoscope was introduced, and the vesical walls were seen to be in the main healthy, with a depression posteriorly. The trigone on the left side was smooth and healthy, with the left ureteral orifice readily discerned and quite normal. In relation to the right ureteral orifice there was a localized area of pronounced bullous œdema and congestion. Opaque ureteral catheters were next introduced. On the left side the catheter easily advanced for a distance of 30 cm. On the right side the ureteral orifice was found with difficulty, and a catheter passed up it was definitely arrested at a distance of 15 cm. From the right catheter a small amount of blood-stained secretion was obtained; from the left the efflux was more copious, a little turbid, but not blood-stained. Specimens of secretion from both sides were collected.

Radiograms were then taken with the ureteral catheters *in situ*. The left



catheter was found to pursue a normal course up to the level of the 3rd lumbar vertebra. The right catheter was seen to be arrested at the level of the brim of the true pelvis; there was no evidence of calculus at this point (Fig. 423).

Twenty c.c. of 20 per cent sodium bromide solution was next injected into the right ureteral catheter without causing discomfort. The resulting ureterogram demonstrated a definite ureteral obstruction, some distance above the tip of the catheter, at the level of the lumbosacral articulation. At this point the ureterogram ended as a small sphere. A very faint shadow of bromide solution was observed in the renal pelvis, situated lower than usual, at the level of the 3rd lumbar vertebra (Fig. 424).

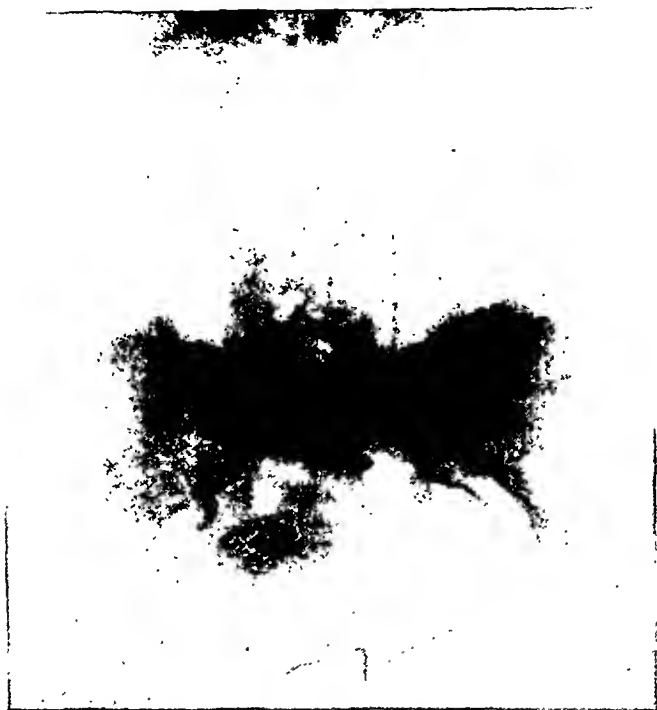


FIG. 424.—Prior to operation. Right-sided ureterogram after injection of a solution of sodium bromide. There is definite ureteral obstruction at the level of the lumbosacral articulation.

#### EXAMINATION OF URINE.—

*Direct Films.*—The left ureteral specimen contained numerous transitional epithelial cells but no organisms. The right ureteral specimen contained numerous transitional epithelial cells and red blood-cells. There were no cells suggesting malignancy, and no organisms were found. The bladder specimen contained red cells, a few epithelial cells, scanty polymorphonuclear leucocytes, and a few Gram-negative bacilli.

*Culture.*—On culture a growth of *Bacillus coli communis* was obtained from the bladder specimen, but no growth resulted from either of the ureteral specimens.

*DIAGNOSIS.*—From the symptomatology and investigation findings a diagnosis was made of tumour affecting the right ureter at the level of the 5th lumbar vertebra, probably originating as a primary growth in the renal pelvis, though possibly primarily situated in the ureter. Operation was advised.

OPERATION.—The patient was operated upon by Professor Wilkie on Nov. 27. Through the usual oblique loin incision the kidney, which was low and mobile, was exposed and easily delivered into the wound. The kidney was a little below the average size, and, apart from a small subcapsular cyst on its anterior aspect, it exhibited no abnormality. The renal pelvis was not dilated, and palpation revealed nothing to suggest a tumour in its interior. The ureter was followed downwards, and its upper 6 cm. was found to be healthy and of small calibre. Just below this level there was a fusiform swelling, measuring about 3 cm. in length, soft in consistency and yellowish blue in colour. There were no adhesions to the surrounding tissues, the outer coat of the ureter being perfectly intact. Below the level of this swelling the ureter again appeared perfectly healthy. No enlarged lymphatic glands were found.



FIG. 425.—Subsequent to operation. Radiogram with opaque catheter introduced into right ureter. The catheter passes up to nearly the same level as before operation.

In view of the patient's age and general condition, and also because the urological investigation had revealed an apparently normal ureter below this level, a complete dissection down to the vesical wall was not carried out. The ureter was divided a short distance below the tumour, its walls being examined and found to be healthy. The vessels of the renal pedicle were then ligated and divided, and the kidney with the portion of ureter attached was removed.

The patient made an uninterrupted recovery. No difficulty was experienced with micturition, and no blood, pus, or albumin appeared in the urine. She was discharged on Dec. 18, three weeks subsequent to her operation.

On April 9, 1925, she reported for examination. She stated that all her urinary

symptoms had been relieved. There had been no haematuria nor frequency, and her urine was now voided easily and without discomfort. She complained of occasional slight pain over the right iliac crest in the distribution of the gluteal branch of the last dorsal nerve. There had been no loss of weight.

On cystoscopic examination the bladder was found to be entirely healthy and of normal capacity. The bullous cystitis round the right ureter had quite disappeared, leaving a small orifice surrounded by flat, pale mucosa, in which a few slightly engorged blood-vessels coursed. A catheter passed up the right ureter with ease for a distance of 10.5 cm., where it was arrested. An X-ray photograph showed the catheter curving round from the beak of the cystoscope, and having its tip at a level a little below the brim of the true pelvis (*Fig. 425*). Six c.c. of sodium iodide solution was injected, and the resulting ureterogram demonstrated that the ureter filled evenly for a further 3 or 4 cm. The ureteral calibre was small, and there was no distortion or filling defect (*Fig. 426*).

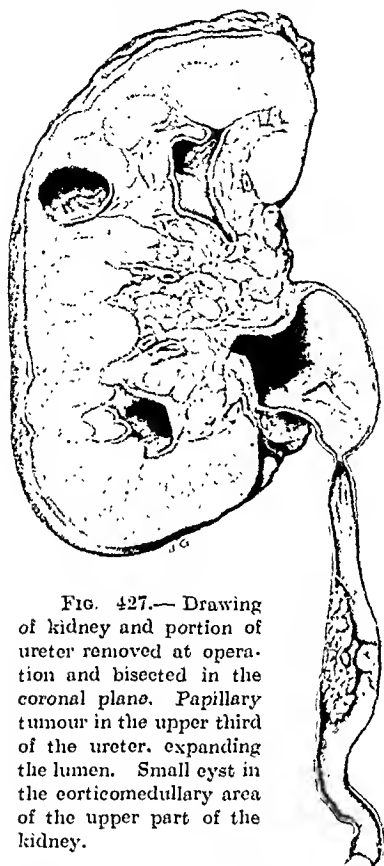


FIG. 426.—Subsequent to operation. Right-sided ureterogram after injection of a solution of sodium iodide. The portion of ureter remaining is filling evenly throughout its course.

The urine was clear, acid in reaction, with a specific gravity of 1.006, and contained no abnormal constituents. Microscopically, one or two epithelial cells only were found; no organisms were obtained either on film or on culture.

**PATHOLOGICAL FINDINGS.**—The specimen was injected and fixed in Pick's solution. It was found that the amount of fluid required to fill the renal pelvis was 6.5 c.c., and that the ureteral lumen at the site of the tumour was so encroached upon as only to admit a very fine probe. After fixation, during the course of which a certain degree of contraction of the tissues took place, the whole specimen was bisected in the coronal plane.

*Macroscopic Appearances (Fig. 427).*—The kidney was small, and measured 9 cm. in length by 5 cm. in breadth. The cortex was narrow, and contained a small cyst 1 cm. in diameter, lying adjacent to the medulla, in the upper portion of the organ. There was an increase of peripelvic fat, but the pelvis itself was not dilated, its mucosa was healthy, and there was a well-marked constriction at the uretero-pelvic junction.



The upper 2 cm. of the ureter was narrow, 4 mm. in diameter, and appeared quite healthy. Below this, for a further distance of 2 cm., the ureter was dilated in a fusiform manner to over twice its diameter above, by a sessile papillary tumour, from which a pedunculated expanded growth was prolonged downwards into the lumen of the channel. The base of the tumour covered practically three-fourths of the circumference of the ureter and almost completely obstructed it. Externally the ureteral wall was somewhat congested, smooth and regular, and exhibited no induration. Immediately distal to the tumour the ureter regained its small calibre and healthy appearance, forming an S-shaped bend at a slightly lower level.

*Microscopic Appearances.*—In order to demonstrate the tumour in its entirety, the anterior half was utilized for complete longitudinal sections. These show the sessile nature of the proximal portion, while distally the tumour is prolonged as a fibrocellular, pedunculated growth, with normal ureteral wall on either side of it (Fig. 428).

Under higher magnification the distal portion evinced the characteristics of a proliferative type of benign papilloma, with hæmorrhagic extravasations towards its periphery, and at one point the formation of a cyst in the centre of an epithelial mass. At the

growing margin (Fig. 429) the loose vascular, ramifying, connective tissue of the

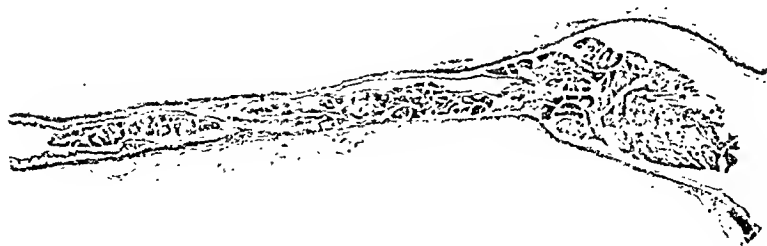


FIG. 428.—Longitudinal section of the tumour under low power. The tumour is sessile in its proximal portion and pedunculated distally. ( $\times 3$ )

neoplasm is seen to be continuous with the ureteral submucosa, and at this point there is a round-celled infiltration. Surmounting the stroma, and sharply demarcated from



FIG. 429.—Section at the attachment of the distal pedunculated portion of the tumour to the ureteral wall. There is an inflammatory-celled infiltration of the submucous coat of the ureter at the growing margin of the tumour.

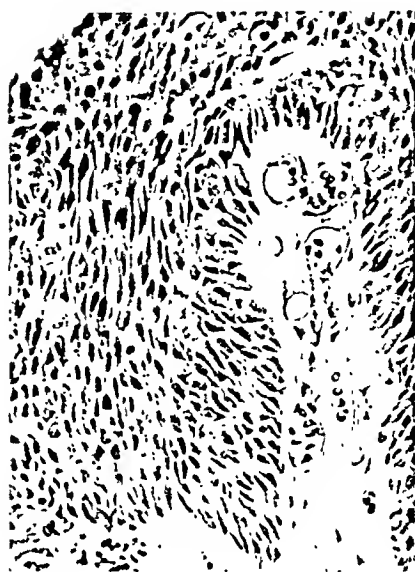


FIG. 430. Drawing of a section of the distal papillomatous portion of the tumour under high power. The individual characteristics of the tumour cells and their relationship to the stroma are seen.



FIG. 431.—Sessile proximal portion of the tumour. Invasion of the submucous coat of the ureter by two islands of tumour-cells, with a surrounding inflammatory-celled infiltration. The intact muscle coat is seen below.



FIG. 432.—Sessile proximal portion of the tumour. Irregular strands of tumour-cells infiltrating the submucosa. A group of dilated blood-vessels in the ureteral adventitia is separated from the tumour by the intact muscle layer.

it. are the epithelial elements, several layers in depth. In the stratum adjacent to the stroma, the epithelial cells are elongated, whereas superficially they are flattened. In the intermediate area more variation occurs; thus, in the denser portions the cells are compressed and tend to be spindle-shaped, while in the looser portions they tend to be cuboidal or polyhedral. Individually the nuclei differ in shape and size, in accordance with the type of cell, and also show variations in their staining properties, some staining much deeper than others, while in a few there is a reticular formation of the chromatin. The cytoplasm is faintly granular and takes up the basophil stain poorly (*Fig. 430*).

The sessile proximal portion of the tumour, while still retaining its papillary nature, showed in places evidences of commencing infiltration, though at no point could this be found to penetrate beyond the submucous layer. The ureteral sub-

mucosa is invaded (*Fig. 431*) by two islands of tumour-cells, surrounded by an inflammatory cell reaction, the intact muscularis being seen immediately below. Similarly the submucous connective tissue, covered by more or less normal mucous membrane, is shown infiltrated (*Fig. 432*) by irregular strands of tumour-cells, some of which tend to be of an undifferentiated type. Again the process stops short of the muscle layer, and a group of dilated blood-vessels is seen in the ureteral sheath externally. Vacuolation and cyst formation were very definite features of this portion of the tumour. A large cyst is depicted (*Fig. 433*), lined by flattened cells, and with two ingrowths, the superficial cells of which are cedematous, swollen, and becoming detached. In the interior of the cyst are some swollen, degenerated tumour-cells and also a few erythrocytes.

Sections taken from the kidney revealed a moderate degree of chronic interstitial nephritis, commensurate

with the age of the patient. The renal pelvis and ureter above the tumour showed no abnormality, but in the portion of ureter situated below the tumour there was slight congestion and an extensive round-celled infiltration of the submucosa and of the perivascular areas in the outer coat.

**Conclusion.**—Primary papillary epithelial tumour of the ureter, with cyst formation and evidences of early malignancy of a local character.

## ETIOLOGY.

Mechanical irritation being such an important etiological factor in the production of tumour growth, it might be expected that ureteral neoplasms would be found existing as sequelæ to calculus formation. In 11 out of 54 cases calculi have been present. In certain instances, however, their etiological significance may be questioned, as it appears quite possible that they may have developed subsequent to the ureteral obstruction and urinary decomposition occasioned by the tumour, or that they have formed in the centre of a blood-clot enclosed by tumour growth. The latter possibility is

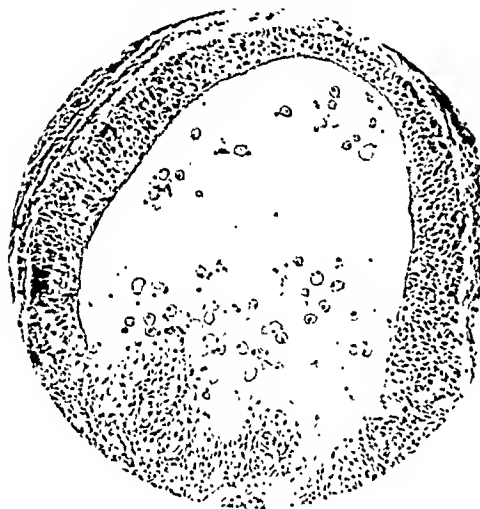


FIG. 433.—Sessile proximal portion of the tumour. Cyst in the centre of the growth, containing swollen, degenerated tumour-cells and red blood-corpuscles.

illustrated in Le Dentu's<sup>2</sup> case, where papillomatous growths were situated at the two extremities of the ureter, each containing a little mass of blood-clot in the centre of which was a small concretion the size of a hemp-seed. Occasionally, however, the constant irritation of an impacted calculus appears undoubtedly to have been of importance as a predisposing factor. In a case published by Metcalf and Safford<sup>3</sup> the patient had suffered for some years from attacks of renal colic and pyuria; he subsequently developed, at the lower end of the ureter, an adenocarcinoma surrounding an impacted oxalate stone. A very similar case was recorded by Davy.<sup>1</sup> In Aschner's case a carcinoma of the upper end of the ureter was associated with a state of leucoplakia, infection, and calculus formation in the renal pelvis.

Calculus formation, apparently, is more frequently associated with malignant than with benign ureteral tumours.

Concomitant anomalies in the ureter have been noted on two occasions. In Neelsen's<sup>5</sup> case multiple papillomatous tumours grew in the upper end of a bifid ureter, whilst in Jona's<sup>6</sup> case a 'papillary epithelioma' occupied a diverticulum at the lower end of the ureter.

**Age and Sex Incidence.**—Neoplasms of the ureter, like those situated elsewhere in the urinary tract, occur most frequently late in life, the average age being in the sixth decade for all forms of tumour. Héresco<sup>7</sup> and Thornton<sup>8</sup> recorded the cases of patients 32 years of age, both with benign tumours, at the lower and upper ends of the ureter respectively; whilst, at the other end of the scale, Richter's<sup>9</sup> case was that of a woman, age 80, with a papillary carcinoma 3 cm. above the ureteral orifice. The influence of sex appears to be of little or no importance as an etiological factor. Of 54 patients, 25 were females and 29 were males, a disparity insufficient in extent to draw conclusions from.

## PATHOLOGY.

The several varieties of ureteral tumour recorded by different observers suggest the following as a simple pathological classification:—

- |                              |                               |
|------------------------------|-------------------------------|
| A. Connective-tissue tumours | Sarcoma.                      |
| B. Epithelial tumours        | { 1. Benign papilloma         |
|                              | { 2. Papillary carcinoma      |
|                              | { 3. Non-papillary carcinoma. |

**Sarcoma.**—This variety of tumour is extremely rare. I have been able to find records of only 5 cases in the literature. Of these, Targett<sup>10</sup> reported on a male, 45 years of age, in whom the whole of the abdominal portion of the ureter was the seat of tumour growth. Willentzki<sup>11</sup> referred to a sarcoma of the upper ureter, and Ribbert<sup>12</sup> observed a myosarcoma spreading from the renal pelvis into the ureter. It is probable that in all these cases the ureter was invaded secondarily.

**Benign Papilloma.**—This type of neoplasm appears to be that most commonly met with. It may be sessile or pedunculated, sometimes markedly so. Culver<sup>13</sup> reported a case in which the main tumour consisted of one long villus, 3 cm. in length, growing from a narrow pedicle and projecting distally.

Papillomata may occur singly, as isolated multiple growths, or as a diffuse papillomatosis, affecting a considerable portion of the ureter. Situated anywhere in the ureter, they show a predilection for either the upper or the lower end; sometimes, as in Le Dentu's case, isolated tumours may occupy both these portions. Should they occur at the lower end, they may be extruded through the ureteric orifice, and secondary tumours may be engrafted on the neighbouring vesical wall. In this manner they may simulate the more common multiple papillomata which, having their origin in the renal pelvis, tend to spread down the ureter, either by direct continuity or by the formation of isolated tumours, and eventually involve the vesical mucosa. A typical example of this latter type of tumour is afforded by a case described by Marion,<sup>14</sup> where a total ureterectomy for diffuse papillomatosis was necessitated five years after a nephrectomy for multiple papillomata of the renal pelvis, and where, in the intervening period, frequent fulguration had been required for recurring papillomata situated in the corresponding side of the bladder.

Histologically, ureteral papillomata are similar in every way to those occurring in the renal pelvis or the bladder. Thus they are formed by a branching vascular stroma, surmounted by multiple layers of transitional epithelial cells, sharply delineated from the underlying connective-tissue core. In the connective tissue, especially at the growing margin, are often found groups of small round cells, probably a manifestation of the irritation produced by the advance of the tumour growth.

**Papillary Carcinoma.**—Papillary tumours of the ureter exhibit a striking tendency towards malignancy. Where a careful histological examination has been carried out on tumours having the macroscopic appearances of benign papillomata, evidences of carcinomatous change have been forthcoming in a number of cases. In some instances the tumour has been found to be exhibiting only early signs of local malignancy, such as increased proliferation of atypical or basal cells with loss of polarity; in others, typical destruction infiltration of the ureteral wall and invasion by tumour-cells of the peri-ureteral tissues have indicated the growth to be essentially carcinomatous. It is important that the whole tumour should be examined, as sometimes only a small portion may show malignant characteristics, the remainder appearing benign. In Richter's case, a localized papillary tumour in the lower third of the right ureter had, in the main, the usual structure of a benign papilloma, except for some variation in type of the epithelial elements. In the central section of the growth, however, tumour tissue in alveolar structure penetrated the muscularis into the connective-tissue sheath, and an infiltrated retroperitoneal gland was found at the autopsy. Almost without exception this type of tumour has formed a single growth and has been situated in the lower portion of the ureter.

**Non-papillary Carcinoma.**—This group comprises the rarest forms of epithelial tumours found in the ureter. Most have been described as medullary or encephaloid, but there are five cases on record of squamous-celled carcinomata. Amongst the latter is Aschner's case, already referred to, in which leukoplakia featured. As is to be expected with so malignant a form of tumour, early extension to surrounding structures has been manifest.



Thus, in Davy's case a mass involving the base of the bladder, and ulcerating into the rectum, had originated in an encephaloid carcinoma of the lower end of the ureter. In Heektoen's<sup>15</sup> case a medullary carcinoma eroded the right iliac bone, and grew to form a mass in the right inguinal region, thereby justifying the clinical diagnosis of osteosarcoma.

**Effects upon the Proximal Ureter and Renal Pelvis.**—Practically all ureteral growths tend sooner or later to produce mechanical obstruction, with the result that the proximal portion of the ureter and the renal pelvis dilate. Secondary hydronephrosis is therefore a common concomitant lesion, and may be extreme if the obstruction is of gradual onset and long standing, as occasioned by the more benign forms of tumour.

**Metastases.**—All forms of malignant tumours are noteworthy for the early metastatic formations occasioned by them in distant organs. Spread may be either by way of the blood-stream, or by lymphatics to the retro-peritoneal lymphatic glands. The liver was found to be the seat of carcinomatous deposits by Voelcker<sup>16</sup> in a case of papillary carcinoma of the lower left ureter; and by Davy, Schmitt,<sup>17</sup> Rundle,<sup>18</sup> and Vorpahl<sup>19</sup> in cases of non-papillary carcinoma. In Rundle and Vorpahl's patients the lungs were also invaded. Spinal metastases were noted in two cases; in Adler's<sup>20</sup> the 4th lumbar vertebra was involved. Gerstein<sup>21</sup> found a small carcinomatous nodule in the right kidney, which he considered secondary on account of an old-standing hydronephrosis and a relatively large carcinoma at the lower end of the corresponding ureter.

### SYMPTOMATOLOGY.

Ureteral tumours possess no pathognomonic symptoms, and thus may simulate such other conditions as renal or vesical tumours, renal calculus, hydronephrosis, or so-called essential hæmaturia.

**Hæmaturia.**—Undoubtedly the commonest symptom is hæmaturia, which is usually the initial sign and may be the only one present throughout the disease. It has been observed in 65 per cent of all cases, and in over 75 per cent of papillomata and papillary carcinomata. The hæmorrhage is usually intermittent, and may be profuse even from a small growth, thus producing a severe degree of secondary anæmia. In a case described by Hofmann<sup>22</sup> the bleeding occasioned by a papilloma the size of a bean was sufficiently copious to cause the death of the patient. As in renal neoplasms, the blood is intimately mixed with the urine, but clots are liable to form, and their passage may excite a typical ureteral colic. Such clots are usually worm-shaped, and portions of them may be so firm as to be mistaken for calculi by the patient, as occurred in Judd and Struthers'<sup>23</sup> case.

**Pain.**—Pain is a less common symptom. For convenience it may be divided into three main types which, of course, may merge into one another: (1) Acute colic from the passage of clots; (2) A more constant ache in the loin from backward pressure and dilatation of the renal pelvis; (3) Severe and sometimes lancinating pain from the pressure on important surrounding structures of an infiltrating malignant growth. The presence of a calculus in association with the tumour may be the cause of varying degrees of pain,

quite apart from that occasioned by the tumour itself. Severe pain of unusual origin was observed in Héresco's case, where a male, 32 years of age, with a benign papilloma at the lower end of the ureter, had suffered for ten years from occasional attacks of renal colic on the same side as the lesion, accompanied by phosphaturia. Occasional subsidiary symptoms, such as slight twingeing pains on the affected side and frequency of micturition, may be explained possibly by the irritation and reaction occasioned by the presence of the tumour, or on the other hand may be due to a coincident urinary infection.

**Hydronephrosis.**—As an objective symptom, hydronephrosis, in varying degree, has been observed in approximately 55 per cent of cases; and in a small proportion, indeed, it has been the sole evidence present of urinary disease. In 1921 Thomson-Walker<sup>24</sup> reported such a case where the diagnosis of diffuse papillomatosis of the ureter was arrived at during an operation for an otherwise symptomless hydronephrosis of eight months' duration in a man who had experienced one attack of hæmaturia four years previously.

It is extremely exceptional that the actual tumour can be palpated or seen externally, apart from where it produces profuse infiltration and local extension. Quinby,<sup>25</sup> however, described the case of a patient where a ureteral tumour was felt as a distinct mass, oval in shape, and situated a little below and to the left of the umbilicus. This tumour was reported on as a mesothelioma of renal *anlage* origin, showing local invasion.

## DIAGNOSIS.

As ureteral tumours may simulate in their symptomatology many other lesions of the urinary tract, diagnosis must rest to a large extent upon the data obtained from a complete urological investigation. Cystoscopy and ureteral catheterization should be combined with a routine radiological examination and pyelo-ureterography, whilst the urine obtained from the bladder and separately from the kidneys should be examined bacteriologically, cytologically, and chemically.

The difficulties of diagnosis may be gauged to some extent from the fact that in nearly 40 per cent of the recorded cases the tumour was discovered post mortem. In several of the clinical cases the correct diagnosis has been arrived at only after one or more previous operations on the kidney, undertaken on the assumption that there was present a tumour or simple hydronephrosis of that organ.

Under two conditions diagnosis may be fairly simple. The first is where a tumour is seen actually projecting from the mouth of the ureter. The second is where bleeding continues after the extirpation of a tumour at or near the ureteral orifice, and on cystoscopic examination blood is seen issuing from that aperture. In the latter case, ureteral catheterization will frequently determine the location of the tumour which is causing the bleeding. In either case, however, it may be impossible to identify the upper limit of the growth, or to ascertain whether the tumour is primarily ureteral or originating in the renal pelvis. The first correct pre-operative diagnosis was probably

made in 1899 by Albarran, in a patient upon whom Le Dentu had previously performed a nephrotomy for renal colic. As the urinary fistula continued to discharge, and as the ureter was found to be obstructed from above, Albarran carried out a cystoscopic examination, discovered a papilloma projecting from the ureteral orifice, and demonstrated the ureteral obstruction at a distance of 5 mm. by means of a catheter.

Where the tumour is situated some distance above the ureteric orifice, accurate diagnosis may be extremely difficult. The cystoscopic findings in such cases may be entirely negative, or blood may be seen emanating from the ureter in jets, as an almost continuous stream, or as a solid cylindrical coagulum. Occasionally the ureteral orifice itself is slightly deformed or shows blood-staining of the mucosa surrounding it. Kraft<sup>26</sup> observed during cystoscopy that direct abdominal pressure over the site of a vascular tumour caused a stream of bright blood to be ejected from the ureter, where before the efflux had been clear even though pressure had been exerted over the kidney above. Renal function as tested by excretion of indigo-carmin has been found to be impaired or lost in some cases, whereas in others it has been equal to that of the healthy side, indicating that the tumour has not caused marked obstruction to the urinary outflow.

Ureteral catheterization may give valuable information in various ways. Where blood is issuing from a ureter, the catheter may be suddenly arrested by a definite obstruction, and if, as was pointed out by Marion, the catheter can be introduced beyond this level, clear urine may be obtained, or hæmatonephrosis, due to central bleeding, may be tapped. Beer<sup>27</sup> obtained no excretion of dye from one side, but a continuous flow of blood occurred after the withdrawal of the catheter, which had been arrested half-way up the ureter. Chiari<sup>28</sup> likewise found an absence of secretion associated with a ureteral obstruction, which bled on impact with the catheter. It is, of course, not uncommon even in a healthy ureter for the tip of a catheter to be arrested in its ascent by a fold of mucous membrane, and also for it to cause traumatic bleeding. Such bleeding, however, is not copious, and there is generally a gradual increase in amount from the moment of its inception, the secretion being clear up to that point. Other sources of ureteral obstruction and possibly of bleeding might be cited, such as simple or tuberculous stricture, calculus, and stenosis of extra-ureteric origin, the last perhaps being most frequently occasioned by the presence of calcified retroperitoneal glands causing pressure on or kinking of the ureter. Any of these conditions may simulate a tumour, and it is therefore very important that the ureteral catheter be made opaque to X rays, and after its introduction a radiogram be taken. In this way the exact site of any form of obstruction can be located, the relationship of the catheter to abnormal shadows ascertained, and the differential diagnosis facilitated.

Pyelo-ureterography has been employed in a small number of the published cases. It is obvious that this method of diagnosis is especially indicated in ureteral tumours, and interesting results have accrued from its use. Thus Quinby found "a definite distortion and obstruction in the ureter, which was curved upward, slightly dilated, and the shadow ended in an abrupt tip opposite the iliac crest". The ureterogram in Kretschmer's<sup>29</sup> case demonstrated

a distorted ureter, which ascended in an S-shaped curve and terminated with its tip overlying the mid-point of the iliac crest. Culver's case was one in which the kidney had been removed previously on account of the patient experiencing repeated hæmorrhages. As the bleeding recurred, ureterography was carried out, but in this instance the ureterogram failed to show any tortuosity, dilatation, or filling defect, and it extended above the site of the tumour, which was an attenuated papilloma not causing ureteral obstruction.

Occasionally the cytological examination of the urine gives valuable information. Thus Richter found atypical epithelial cells in his case, and Kraft discovered definite detached papillomatous villi. Needless to say, the mere finding of transitional epithelium, even in considerable amount, is of no import. Similarly the presence of pus-cells and organisms does not negative tumour, superimposed infection being not uncommon.

### TREATMENT.

The surgical treatment of ureteral tumours depends upon their pathology, upon their site and extent, and upon the general condition of the patient. In those who are poor surgical risks from old age, excessive hæmorrhage, or other grave complications, modifications will be found necessary in the operative technique employed.

In most cases the ideal treatment is complete nephro-ureterectomy, carried out in either one or two stages. Where the growth involves the ureteral orifice or surrounding bladder wall, a partial cystectomy may require to be added, though satisfactory results have been obtained from fulguration of the vesical extension prior to the major operation. Such a drastic form of treatment is indicated on three main grounds: (1) The kidney is generally hydronephrotic, otherwise diseased, or will become functionless as a result of the operation. (2) It is frequently difficult to gauge the exact extent of the tumour; widespread extensions along the ureter may occur, either in continuity or separated by considerable areas of healthy mucosa. (3) Although apparently benign, these tumours may be actually malignant or in a state of incipient malignancy. The complete operation was first performed by Albarran and Le Dentu in 1899.

If a single tumour involves the upper portion of the ureter, and if it has been established previously that the remainder of the ureter is healthy, then nephrectomy and ureterectomy to a point well below the growth is justified. Such patients should report at regular intervals afterwards for examination.

In those tumours of the lower end of the ureter which project into the bladder as pedunculated papillomata, endoscopic fulguration or local excision with re-implantation of the ureteral stump into the bladder may, in certain cases, be the method of choice. The latter form of treatment was first employed in 1899 by Léonté,<sup>30</sup> as reported by Héreseo, and subsequently by Albarran,<sup>31</sup> Brunet,<sup>32</sup> Mackenroth,<sup>33</sup> and Finsterer.<sup>34</sup> In each case recovery ensued, but in the first mentioned an acute pyelonephritis followed the operation and necessitated a nephrectomy.

Marion<sup>35</sup> records two cases treated by endoscopic fulguration. In the first a vesical papilloma in the region of the right ureter was so treated in a female

of 66 years. Hemorrhage recurred, and a tumour situated 5 cm. above the ureteral orifice was similarly dealt with, a successful result being obtained. In his other case a second tumour was likewise situated 5 cm. above the lower end of the ureter, and clear urine was evacuated by means of a ureteral catheter passed beyond it. Following upon fulguration, however, a condition of hæmatonephrosis developed, a nephrectomy was performed, and the cause of the bleeding was discovered to be a series of multiple papillomata in the renal pelvis.

It is evident that operative measures directed only towards papillary tumours at the lower end of the ureter tend to be incomplete. They do not take into account the condition of the kidney and ureter above, which may well be the site of a primary growth. These forms of treatment should be undertaken therefore only in the case of those who are bad surgical risks, as the first step in a deliberate two-stage operation, or on the understanding that further operative interference may be required at a future date.

### SUMMARY.

1. Primary tumours of the ureter are rare, and are met with chiefly in the sixth decade of life.

2. Calculus is not a common predisposing factor; when present it is generally associated with malignant forms of tumour.

3. Pathologically these tumours are most frequently papillary in type and many show the structure of benign papillomata, in which case they may be multiple or diffuse. There is a decided tendency towards malignancy—papillary carcinomata, medullary carcinomata, and squamous epitheliomata occur. Sarcomata are excessively rare.

4. Any portion of the ureter may be involved, although there is evidently a predilection towards either the upper or the lower extremity.

5. The symptoms produced are not pathognomonic of the disease, and are the same as those engendered by renal neoplasms—namely, hæmaturia, pain, and sometimes renal enlargement due to the tumour obstructing the ureter and causing hydronephrosis.

6. Accurate diagnosis is usually very difficult, but considerable aid is obtained from the employment of ureteral catheterization and pyelo-ureterography. It is important to locate the downward extent of the tumour, with special reference to the possibility of multiple growths.

7. Radical operation is indicated in treatment. In most instances, nephro-ureterectomy, either complete or partial, is the method of choice. Fulguration of tumours situated at the lower end of the ureter has given encouraging results in patients unsuited for the more radical surgical measures.

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## SUBPHRENIC ABSCESS.\*

BY LIONEL R. FIFIELD AND R. J. McNEILL LOVE, LONDON.

This condition was described by Barlow as long ago as 1845, and about 1873-4 Hilton Fagge published a paper on "Abscess within the Upper Part of the Abdomen". The first operation for subphrenic abscess, however, was recorded by von Volkmann in 1879. Thenceforth numerous additions to the literature were made in England, America, Germany, and Italy, and in 1908 Barnard's comprehensive paper appeared. It was based on a study of 76 cases at the London Hospital, and contained, among other things, a very complete description for practical purposes of the anatomical boundaries of the subphrenic spaces. It is essential to be able to visualize these if the causes and sequelæ of subphrenic suppuration are to be clearly understood and correct treatment is to be instituted.

## ANATOMY AND ETIOLOGY.

The abdominal surface of the diaphragm (*Fig. 134*) is covered by peritoneum except in one place, where it is in contact with the 'bare area' of the liver (in reality it is separated from this by cellular tissue). This is known as the *right extraperitoneal subphrenic space*, and is bounded anteriorly by the right lobe of the liver and right suprarenal body, posteriorly by the diaphragm, to the left by the inferior vena cava and right crus of the diaphragm, above by the anterior layer of the coronary ligament, below by the posterior layer, and to the right by the meeting of these two layers to form the right lateral ligament. Anteriorly its cellular tissue is continuous over the top of the liver with that in the falciform ligament, inferiorly with that behind the ascending colon and right lumbar fossa, and to the right with that in the right lateral ligament, whilst it is continuous round the arcuate ligaments with the cellular tissue in the thorax.

The formation of an abscess in this space has resulted from infection from the liver and biliary passages; from the abdominal wall by way of the lymphatics round the epigastric vessels, which are continuous with those in the falciform ligament; from the right retroperitoneal tissue following perforation of a posterior duodenal ulcer, retrocæcal appendicitis, ulceration of the ascending colon and hepatic flexure, a high perinephric abscess, and pancreatic suppuration; and from supradiaphragmatic conditions such as empyema, bronchiectasis, and abscess of the lung.

In the present series of cases there were 20 right extraperitoneal subdiaphragmatic abscesses, of which 12 followed appendicitis, 1 perforation of a duodenal ulcer, 1 tuberculous periosteomyelitis of a rib, 2 pyonephrosis (1 being calculous in origin, the other tuberculous), and 4 were of doubtful origin.

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\* Based upon 78 consecutive cases occurring at the London Hospital.

There is a tendency for suppuration in this space to spread forward over the top of the liver between the two layers of the falciform ligament, and for an abscess to form in the epigastrium. This happened in 2 of the present series of cases, an abscess forming and being opened a little below the ensiform process. This sequel suggests that when a right extraperitoneal abscess is suspected the epigastrium should be carefully palpated. If fullness be detected, the possibility of anterior drainage between the two layers of the falciform ligament should be carefully considered, which, if practicable, might obviate transpleural drainage.

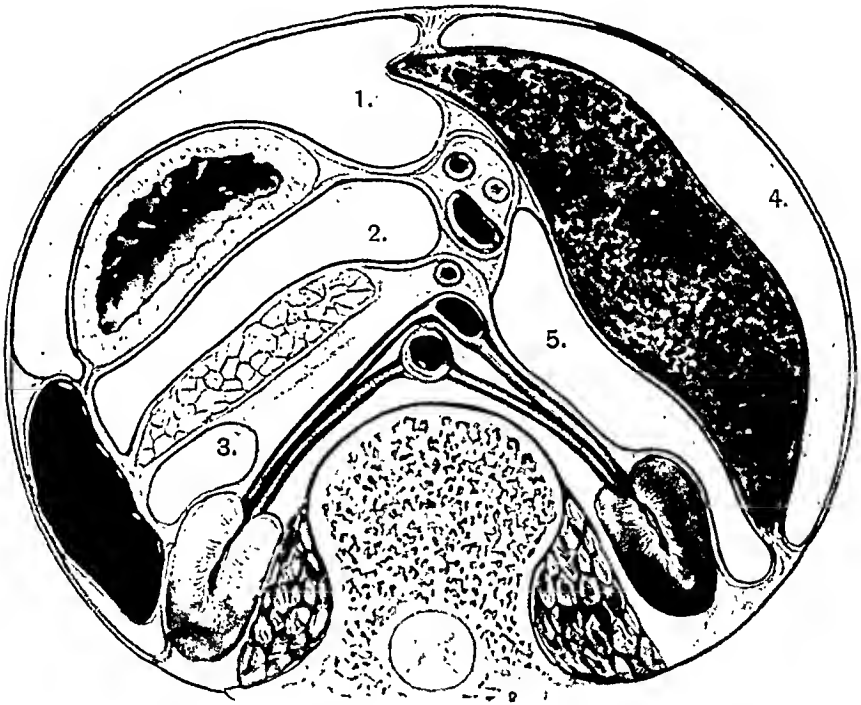


FIG. 434.—Diagram showing anatomical relationships of five types of subphrenic abscess. 1, Left anterior intraperitoneal; 2, Left posterior intraperitoneal; 3, Left extraperitoneal; 4, Right anterior intraperitoneal; 5, Right posterior intraperitoneal.

Anatomically the *left extraperitoneal subphrenic space* does not exist. When a left extraperitoneal abscess forms, it commences round the upper pole of the left kidney and extends upwards, stripping the peritoneum off the diaphragm. It is then bounded above by the diaphragm; behind by the upper part of the left kidney and suprarenal; anteriorly by the end of the œsophagus, the bare area of the stomach, and below this by the lesser sac, pancreas, and spleen; to the left by the diaphragm; and to the right by the aorta and vertebral column. A left extraperitoneal abscess has resulted from perforation of a posterior gastric ulcer, the lesser sac having been obliterated by adhesions (Barnard, Mayo Robson); left retroperitoneal inflammation,



including a high perinephric abscess, ulceration of the colon, diverticulitis, acute periosteomyelitis of lumbar vertebrae, perforation of a carcinoma of the lower end of the œsophagus; and supradiaphragmatic conditions mentioned previously.

In the present series of cases there was only 1 left extraperitoneal abscess, and this resulted from perforation of a carcinoma of the lower end of the œsophagus.

In front and above the right extraperitoneal space is the *right anterior intraperitoneal subphrenic space*. This lies between the superior, anterior, and right lateral surfaces of the right lobe of the liver and the corresponding part of the diaphragm. It is limited posteriorly by the anterior layer of the coronary ligament and the right lateral ligament, to the left by the falciform ligament, and is continuous with the right posterior intraperitoneal space round the border of the right lateral ligament and the margin of the liver. It also freely communicates with the paracolic groove or right lumbar fossa lying to the outer side of the ascending colon; this groove communicates with the pelvis round the outer side of the caecum. When an abscess forms in the right anterior subphrenic pouch, it is generally limited below by adhesions between the transverse colon, great omentum, and abdominal wall. If, however, it results from a paracæcal appendicitis or from hepatic suppuration, the anterior border of the liver becomes adherent to the diaphragm and abdominal wall, the lower boundary being thus formed. Right anterior subphrenic abscess has resulted from appendicitis, rupture of a duodenal ulcer or gastric ulcer, and suppuration in the liver and biliary passages.

In the present series of cases this subphrenic space was involved 13 times, 6 following appendicitis, 3 perforation of a duodenal ulcer, 2 perforation of a gastric ulcer, and 2 were doubtful.

Below the right extraperitoneal space, and somewhat anterior to it, is the *right posterior intraperitoneal subphrenic space*. This is often described as Cantlie's subhepatic pouch or Rutherford Morison's kidney pouch. It is pyramidal in shape, and lies transversely beneath the right lobe of the liver. To the right its base is in contact with the right lobe of the liver and the diaphragm, projecting below this somewhat so that it can be reached by an incision below the 12th rib. To the left its apex is situated at the foramen of Winslow, and below this it is bounded medially by the duodenum. Anteriorly are the liver and gall-bladder; posteriorly the upper half of the right kidney and diaphragm; superiorly the liver, posterior layer of the coronary ligament, and the right lateral ligament; and inferiorly the transverse colon and hepatic flexure. It communicates with the right anterior subphrenic space round the right lateral ligament and margin of the liver, with the right lumbar or paracolic groove round the outer side of the hepatic flexure, and with the lesser sac through the foramen of Winslow.

When an abscess is present, it is separated from the left anterior subphrenic space by adhesions between the left lobe of the liver and the anterior surface of the stomach, and it is separated from the general peritoneal cavity by adhesions between the margin of the right lobe of the liver, abdominal wall, transverse colon, and great omentum. The foramen of Winslow is generally closed by adhesions.

Subhepatic abscess follows appendicitis, perforation of a duodenal or gastric ulcer, hepatic or biliary suppuration, operations on the gall-bladder and ducts, duodenum, and stomach, and suppuration above the diaphragm.

In the present series of cases there were 29 subhepatic abscesses, of which 12 resulted from appendicitis, 5 perforation of a duodenal ulcer, 3 acute cholecystitis, 1 perforation of the common duct by a gall-stone, 3 perforation of a gastric ulcer, 1 a gastric operation, 1 a fractured pelvis, and 3 were of doubtful origin. The sequence of events which led to the formation of an abscess after a fracture of the pelvis was as follows: At the time of fracture the membranous urethra was torn; this led to the formation of a peri-urethral abscess, followed by an abscess in the cave of Retzius and one in the right iliac fossa. Retroperitoneal inflammation (cellulitis and lymphangitis) spread upwards behind the ascending colon, and, contrary to what one would have expected (i.e., the formation of a right extraperitoneal abscess), led to the formation of a right posterior intraperitoneal abscess.

The *left anterior intraperitoneal space* is bounded above by the diaphragm; behind by the left lateral ligament and lobe of the liver, gastrohepatic omentum, and the anterior surface of the stomach; below by the adhesion of the stomach or great omentum to the anterior abdominal wall or diaphragm; to the right by the falciform ligament; to the left by the spleen and gastrosplenic omentum and the diaphragm; and anteriorly by the diaphragm and anterior abdominal wall. It communicates to the right with the subhepatic pouch beneath the postero-inferior border of the falciform ligament in front of the pylorus. To the left it leads in front of the phrenico-colic ligament (or sustentaculum lienis) to the left paracolic or lumbar groove, and hence to the pelvis along the outer side of the iliac colon. Immediately to the inner side, and just in front of the splenic flexure, it communicates with a groove between the vertebral column and the descending colon which also leads down to the pelvis.

An abscess in the left anterior subphrenic space is sometimes described as perigastric or perisplenic, and has resulted from perforation of a gastric or duodenal ulcer or carcinoma, abscess of spleen, hepatic suppuration, or pelvic suppuration spreading upwards along the left internal or external lumbar fossa. It may also follow partial gastrectomy or colectomy.

In the present series of cases there were 14 left anterior intraperitoneal subphrenic abscesses; 8 followed operations on the stomach, 3 perforation of gastric ulcer, 2 perforation of duodenal ulcer, and 1 cancer of the stomach.

The *left posterior intraperitoneal subphrenic space* is the lesser sac. It is bounded posteriorly from above downwards by the diaphragm, pancreas, transverse mesocolon, transverse colon, and the two posterior layers of the great omentum; and anteriorly by the spigelian lobe of the liver, gastrohepatic omentum (lesser omentum), stomach, and two anterior layers of the great omentum. To the left it is limited by the lienorenal ligament, spleen and gastrosplenic omentum, and the meeting of the layers of the great omentum; to the right by the meeting of the layers of the great omentum, and by the proximal third of the first part of the duodenum, above which is the foramen of Winslow, through which the lesser communicates with the greater sac, i.e., the right posterior intraperitoneal space.

An abscess of the lesser sac may result from perforation of a posterior gastric ulcer or rarely a duodenal ulcer, pancreatitis, infection of a pseudocyst of the pancreas, and splenic or hepatic suppuration.

In the present series of cases there were 7 abscesses of the lesser sac; 4 resulted from perforation of a posterior gastric ulcer, and 1 chronic perforation or 'leaking' of a gastric ulcer; 1 followed perforation of an ulcer on the posterior wall of the proximal third of the first part of the duodenum (which is a right anterior boundary of the lesser sac), and 1 resulted from acute cholecystitis, being associated with a right posterior intraperitoneal abscess.

**Etiology.**—*Appendicitis* may lead to the formation of a subphrenic abscess in the following ways (*Fig. 435*):—

1. If the appendix is paracæcal in position, inflammation may spread up the right lumbar fossa to the outer surface of the liver and so to the right anterior subphrenic space; or the inflammation on reaching the lower border of the liver may pass round the hepatic flexure into the subhepatic pouch, from which rarely it may spread to the lesser sac by way of the foramen of Winslow, which, however, is usually closed by adhesions before this can occur.

2. When the appendix is retrocæcal in position, inflammation may spread up behind the ascending colon (i.e., an ascending retroperitoneal cellulitis) to the right extraperitoneal subphrenic space.

3. Should the appendix be in the splenic position, i.e., lying behind the lower end of the ileum and mesentery, inflammation may spread by way of the left internal lumbar fossa to the left anterior subphrenic space.

4. A pelvic appendix abscess may extend by way of the left internal or external lumbar fossa to the left anterior subphrenic space.

5. Appendicitis in any position may be followed by pylephlebitis, and the hepatic suppuration lead to the formation of subphrenic abscesses.

In the present series appendicitis was the cause of 30 subphrenic abscesses—12 right extraperitoneal, 12 right posterior intraperitoneal, and 6 right anterior intraperitoneal.

A gastric ulcer may perforate and result in the formation of a subphrenic abscess. The perforation is generally on the anterior surface of the stomach, the left anterior intraperitoneal subphrenic space being infected. If a posterior ulcer rupture, an abscess of the lesser sac may follow; this space, however,

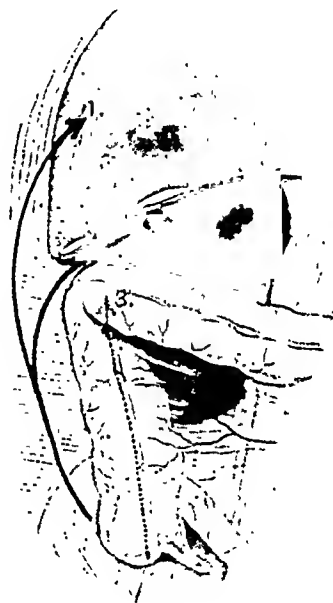


FIG 435. — Diagram showing paths of infection to the subphrenic spaces following acute appendicitis. 1, 2, Right anterior and posterior intraperitoneal respectively (along paracolic groove); 3, Right extraperitoneal (along extraperitoneal cellular tissue).

is often shut off by adhesions, so that the rupture occurs into the cellular tissue round the upper pole of the left kidney, a left extraperitoneal abscess resulting (Mayo Robson, Barnard). Occasionally perforation of a gastric ulcer may lead to a right-sided intraperitoneal subphrenic abscess.

In the present series perforation of a gastric ulcer was the origin of 12 subphrenic abscesses, of which 4 were left posterior intraperitoneal, 3 left anterior intraperitoneal, 3 right posterior intraperitoneal, 2 right anterior intraperitoneal.

Perforation of a *duodenal ulcer* usually produces an abscess in the sub-hepatic pouch, and not infrequently in the right anterior intraperitoneal space ; but perforation posteriorly may lead to a right extraperitoneal subphrenic abscess. The left anterior subphrenic space and the lesser sac may be infected as a result of duodenal perforation.

In the present series subphrenic abscess followed 12 cases of perforated duodenal ulcer, and of these abscesses 5 were right posterior intraperitoneal, 3 right anterior intraperitoneal, 2 left anterior intraperitoneal, 1 was left posterior intraperitoneal, and 1 right extraperitoneal.

*Hepatic suppuration* may be followed by any type of subphrenic abscess, but usually by right extraperitoneal or right anterior intraperitoneal. In this series there was no example of a subphrenic abscess resulting from this cause.

*Suppuration in the biliary passages* may lead to the formation of a subphrenic abscess. In the present series acute cholecystitis was followed by subhepatic abscess in 3 cases, 1 of which was accompanied by an abscess of the lesser sac ; perforation of the common bile-duct by a gall-stone led to the formation of a subhepatic abscess.

CAUSES AND VARIETIES OF SUBPHRENIC ABSCESES.

CAUSE	RIGHT EXTRAPERI- TONEAL	RIGHT ANTERIOR	RIGHT POSTERIOR	LEFT ANTERIOR	LEFT POSTERIOR	LEFT EXTRAPERI- TONEAL
Appendix .. ..	12	6	12	—	—	—
Gall-bladder .. ..	—	—	4	—	1	—
Operations on stomach	—	—	1	8	—	—
Perforated duodenal ulcer	1	3	5	2	1	—
Perforated gastric ulcer ..	—	2	3	3	4	—
Chronic gastric ulcer .. ..	—	—	—	—	1	—
Carcinoma of stomach .. ..	—	—	—	1	—	—
Carcinoma of œsophagus ..	—	—	—	—	—	1
Kidney .. ..	2	—	—	—	—	—
Fractured pelvis .. ..	—	—	1	—	—	—
Rib .. ..	1	—	—	—	—	—
Unknown .. ..	4	2	3	—	—	—
Totals ..	20	13	29	14	7	1

AGE AND SEX INCIDENCE.—

Age (years)	0-10	11-20	21-30	31-40	41-50	51-60	61-70
Cases ..	6	15	19	17	13	6	2
Total ..	78 (43 males, 35 females).						

## PATHOLOGY.

Infection of the subphrenic space may result from:—

1. Wounds.
2. Gravitation of inflammatory exudate from general or local peritonitis or of extravasated contents of a viscus (spontaneous or operative).
3. Hæmatogenous infection.
4. Direct extension from a neighbouring viscus—e.g., rupture of a liver abscess into the right extraperitoneal space.
5. Lymphatic spread. —
  - a. The retroperitoneal lymphatics.
  - b. The diaphragmatic lymphatics. There are supra- and infra-diaphragmatic plexuses intercommunicating freely. If there be pus under pressure above the diaphragm, infection may spread through to the subphrenic spaces, but the extension of infection in the opposite direction is a very much more frequent occurrence.
  - c. The lymphatics along the deep and superior epigastric vessels communicate with those in the falciform ligament.

In connection with (2) it is necessary to consider briefly the *abdominal watersheds*. The posterior abdominal wall presents certain well-marked watersheds: the median longitudinal, formed by the vertebral column; and the transverse, formed by the forward convexity of the lumbar curve, and on each side of this the kidney in its perinephric fat and the thick muscles of the loin. Fluid tends to collect in the subphrenic spaces above and the pelvis below; if the patient be tilted sufficiently (i.e., placed in Fowler's position), it will flow from the former over the transverse watershed round the outer side of the cæcum and iliac colon into the pelvis, where it is much less rapidly absorbed and more easily treated.

The relationship of the pylorus and first part of the duodenum to the median watershed is of much interest. Generally they lie to the right of it, so that right-sided subphrenic abscesses may follow perforation of them. If they lie to the left, left-sided abscesses may result. This was the explanation given by Box of the cases he reported, and it certainly seems satisfactory. Rolleston, in a similar type of case to those of Box, thought that previously-formed adhesions were the main factor in determining the direction of the flow of extravasated material to the left side.

Extraperitoneal subphrenic abscess is an example of cellulitis terminating in suppuration.

Intraperitoneal subphrenic abscess is an end-result of acute localized peritonitis. Inflammatory exudate is poured out, and after ten to fourteen days a localized abscess is present, walled in by fibrin and, later, granulation tissue. The abscess may be uni- or multilocular, and not infrequently two spaces are involved at the same time. In the present series of cases there were 6 examples of this. On two occasions both the right anterior and posterior subphrenic intraperitoneal spaces were involved at the same time following perforation of a duodenal ulcer; in another case involvement of the right anterior intra- and right extra-peritoneal spaces resulted from perforation

of a duodenal ulcer; perforation of a gastric ulcer led to the formation of left and right anterior subphrenic abscesses; purulent cholecystitis with portal pyæmia resulted in left and right posterior intraperitoneal abscesses; and perforation of a gastric ulcer was followed by left anterior and right posterior intraperitoneal abscesses.

The abscess often contains gas, which may have escaped from a perforated viscus or been formed by organisms, e.g., the *Bacillus coli communis*. Fluid present is mainly purulent exudate, the result of bacterial and chemical irritation, but it may also contain gastric and duodenal contents, bile, liver-cells, echinococcal cysts, etc.

As the abscess enlarges, the diaphragm is pushed upwards, obliterating the costophrenic space, which not infrequently becomes closed by adhesions; clear fluid, often in large amount, collects in the pleural cavity, and empyema may follow. The lung is pushed upwards and may be collapsed. In the absence of adhesions, depression of the abdominal viscera may result, especially in the case of extraperitoneal abscess.

Many different kinds of organisms may give rise to subphrenic suppuration; but those most commonly present include the *Bacillus coli communis*, streptococci, staphylococci, etc., as shown in the table below. Tubercle bacilli and actinomyces have been demonstrated. In the present series the pus was examined in 30 cases, with the following results:—

<i>Sterile</i> :—Acute cholecystitis	..	..	..	..	2
Tuberculosis of kidney	..	..	..	..	1
Tuberculosis of rib	..	..	..	..	1
Appendix	..	..	..	..	1
Perforated gastric ulcer	..	..	..	..	1
Doubtful	..	..	..	..	2
					— 8
<i>Staphylococci</i> :—Appendix	..	..	..	..	— 4
<i>Streptococci</i> :—Appendix	..	..	..	..	1
Perforated duodenal ulcer	..	..	..	..	1
					— 2
<i>Pneumococci</i> and <i>Streptococci</i> :—Perforated gastric ulcer	..	..	..	..	— 1
<i>B. Coli</i> and <i>Streptococci</i> :—Appendix	..	..	..	..	— 1
<i>B. Coli</i> :—Appendix	..	..	..	..	2
Doubtful	..	..	..	..	1
					— 3
<i>B. Coli</i> and <i>Staphylococci</i> :—Appendix	..	..	..	..	6
Perforated gastric ulcer	..	..	..	..	1
Operation on stomach	..	..	..	..	2
Doubtful	..	..	..	..	1
					— 10
<i>Friedländer Group</i> :—Appendix	..	..	..	..	— 1
RELATIVE FREQUENCY OF ORGANISMS :— <i>B. coli</i>	..	..	..	..	14
Staphylococci	..	..	..	..	14
Streptococci	..	..	..	..	4
Pneumococci	..	..	..	..	1
Friedländer group	..	..	..	..	1
Sterile	..	..	..	..	8

From a consideration of the anatomy, previously described, of subphrenic abscess, it will be seen that *rupture* may occur into the pleura, lung (and bronchus), pericardium, œsophagus, general peritoneal cavity, and externally. In the present series of cases 5 perforated the diaphragm into the lung (or bronchus), 4 into the pleural cavity only (producing empyema), 1 into the

gall-bladder, 2 into the hepatic flexure of the colon, and one penetrated into the spleen. In no case did rupture into the general peritoneal cavity occur.

Although gastric and duodenal fistulae may follow a subphrenic abscess, they are distinctly uncommon, and no example of this sequela is to be found in the present series.

### DIAGNOSIS.

It has always been recognized that the diagnosis of this condition is occasionally very difficult, as is indicated by the fact that out of this series of 78 cases only 59 were subjected to operation, leaving a residue of 19. A few of these were not suitable for operation owing to the condition of the patient or the nature of the primary lesion: but the majority might have been given the benefit of surgical treatment had the presence of a subphrenic abscess been suspected and the pus sought.

In obscure abdominal cases where symptoms of infection are present it is well to bear in mind the aphorism "Pus somewhere, pus nowhere else, therefore pus under diaphragm"; and many cases are diagnosed only by the exclusion of pus elsewhere and exploration of the subdiaphragmatic spaces.

The history is in most cases suggestive. A typical case is one in which an infective focus in the abdominal cavity has been dealt with, and the condition of the patient temporarily improves, after which the signs and symptoms of toxæmia reappear. The quiescent interval may be as long as four months, as occurred after a case of appendicitis in this series, during which period the patient was at work; but more commonly the primary lesion almost merges into the period during which the subdiaphragmatic infection dominates the clinical picture. As with liver abscesses, the rapid absorption of pus produces marked features of toxæmia; hence irregular temperature, sweating, wasting, and anemia are frequently in evidence, which arouses the suspicion of some obscure focus of infection. Furthermore, the patient may complain of fullness, pain in the upper abdomen, or omalgia.

**Physical Examination.**—Inspection of the chest and abdomen may reveal local abdominal swelling, bulging of the ribs, or immobility of the chest. In 21 of our series of cases a swelling was palpable in the abdomen or loin. In a few cases, particularly with right extraperitoneal abscesses, the liver was displaced downwards; but this is by no means constant even with large collections of pus, as adhesions round the liver may fix it in position, in which case signs in the chest are very definite.

Examination of the chest is of extreme importance. In the majority of cases pressure signs at the base of the lung may be recognized; in other cases pleural effusion or empyema are not uncommonly associated conditions. If gas is present in appreciable quantity, then percussion may yield characteristic information in that four zones of altered resonance are present; thus, below, a dull area is found due to the liver; above this the gas gives a tympanic note; then a zone of diminished resonance indicates pressure on the lung with partial collapse; while above, normal lung resonance may be obtained.

In some recorded cases the presence of Grocco's triangle has been noted on the opposite side, and it has disappeared after drainage of the abscess.

**Accessory Investigations.**—As would be expected, a blood-count usually indicates the presence of pus, provided the patient's resistance is sufficient to stimulate reaction; but the most valuable assistance in localizing the presence of pus is radiographic examination. Out of 37 cases in this series, in which the result of radiographic examinations are recorded, no fewer than 21 were definitely diagnosed, in 9 cases an abscess was suspected, and in the remaining 7 cases no evidence was found. Three abnormalities may be recognized by the screen: (1) Elevation of the diaphragm, which normally on the right side reaches the 5th rib; (2) Diminished mobility, the usual excursion during deep respiration being 1 to 1½ in.; (3) Alteration of contour due to the presence of a local collection of pus. In addition to these features, the presence of gas may give valuable information, as the result in clear areas may be easily detected.

The final court of appeal is the exploring needle, but explorations should always be performed in the theatre, so that drainage may be established at the same time, otherwise infection along the needle track is possible, and subsequent exploration may miss the abscess.

**Differential Diagnosis.**—Owing to the insidious onset, and frequently obscure physical signs, the differential diagnosis of this condition is sometimes difficult.

*Pylephlebitis* is frequently due to the same causes as subphrenic abscess, so that the history of these two conditions may be identical. The onset of pylephlebitis, however, is usually abrupt, possibly ushered in by a rigor, and rigors during the course of this disease are common. Hepatic enlargement is generally obvious, but it may be distinguished from the displaced liver sometimes associated with subphrenic abscess in that tenderness is present. Radiographic evidence is valuable, and later, jaundice may occur in cases of pylephlebitis.

*Empyema* is another condition which closely simulates subphrenic abscess or may be associated with it. The respective histories often indicate which condition is present, and lateral cardiac displacement is typical of fluid in the chest. X-ray appearances, if showing gas or local bulging, may distinguish the two conditions. Frequently the exploring needle must be used, the movement of the needle during respiratory excursions then indicating the subdiaphragmatic site of the abscess.

The physical signs of *liver abscess* so closely simulate subdiaphragmatic infection that distinction may be impossible, although the history of the case or the presence of amœbæ in the stools may be suggestive.

*Perinephric infection* may cause some confusion in diagnosis; but history, routine examination, and site of swelling should establish a diagnosis.

Among other conditions which have been mistaken for subphrenic abscess are aortic aneurysm, pancreatic cysts, and renal tumour; but the non-inflammatory natures and characteristic features of these conditions should distinguish them.

### TREATMENT.

The treatment of subphrenic abscesses is, to a certain extent, *prophylactic*; the adoption of Fowler's position in some cases, and efficient drainage where



a primary infective focus is dealt with, have certainly reduced the incidence. Previous to the general practice of placing the patient in Fowler's position in cases of acute appendicitis with infection, the subsequent formation of a subphrenic abscess was a comparatively common complication; now it is estimated that in only 0.6 per cent to 1 per cent does subphrenic abscess occur. Little can be done to prevent lymphatic infection, but gravity would seem to be of some importance in discouraging pus from tracking upwards towards the diaphragm.

In cases of acute appendicitis the incidence of subphrenic infection is much reduced when the delayed method of treatment is adopted. This treatment gives cases of localized or general peritonitis, i.e., cases in which the appendix is perforated, an opportunity to subside, so that a clean operation may be performed later. In the majority of cases acute symptoms successfully abate and the virulence of the infection subsides as the resistance of the patient increases. Thus, in 228 cases treated on delayed lines at the London Hospital, subphrenic abscess occurred in only one case, whereas in 1109 cases subjected to immediate operation 7 were complicated by subphrenic abscess.

Another point of importance in connection with prophylaxis is the establishment of efficient drainage where such is necessary. Subphrenic abscess appears to be more common in retrocecal and paracolic appendicitis than when the organ points downwards or towards the left. This is doubtless due to infection along the retroperitoneal cellular tissue, and partly to infection passing along the paracolic groove, and hence drainage by a stab wound in the loin tends to counteract these two modes of infection. During the years 1919-22 no case of subphrenic abscess occurred when drainage was established by this route.

Little can be accomplished in the way of prophylaxis regarding cases of perforated ulcer in the upper abdomen, as the harm has already been done before surgical intervention. However, the careful removal of extravasated visceral contents, the adoption of Fowler's position, and drainage if necessary, mitigate against the formation of an abscess. The almost routine use of a drainage tube after cholecystectomy makes this operation a very uncommon cause of subphrenic infection; in this series the 4 cases connected with gall-bladder lesions were already the subject of gross infection.

Regarding the *actual treatment* of the abscess, the search for pus should always be conducted in the theatre. In cases where a lump can actually be palpated in the loin or abdomen, then incision and drainage are adopted. In the latter cases posterior drainage should be established if possible, by means of a stab wound, in which case the anterior incision may be closed.

When needling is indicated the needle should be inserted through the lower intercostal spaces in the posterior axillary line; failing detection of an abscess, puncture should then be made in the mid-axillary and paravertebral lines. For an adult of average size a 4-inch needle of wide bore is used, as pus may be thick. When pus is found, the respiratory movements of the diaphragm are transmitted to the needle, indicating that the abscess is below the diaphragm.

The rib below the needle is excised subperiosteally for about three inches.

If necessary the parietal pleura is stitched to the diaphragm. As a general rule, adhesions have already formed, but too much reliance must not be placed on their presence. Subsequent collapse of the diaphragm when the support of pus is withdrawn may separate adhesions between it and the parietal pleura, allowing secondary infection of the pleural cavity. Hence, in doubtful cases, and especially at a high level, e.g., the 8th rib, sutures provide an extra safeguard. The diaphragm is then incised across its fibres, a finger introduced, and the drainage tube inserted in the cavity.

### PROGNOSIS.

Out of the 78 cases which comprise this series, 39 terminated fatally, with or without operation; that is, exactly 50 per cent. In some of the cases which died before operation doubtless the primary condition was the actual cause of death. Eliminating these, we found that of 59 cases submitted to operation 39 recovered and 20 died, giving a mortality of 32 per cent.

On analysing the results of different operative measures we find as follows:—

OPERATION	RECOVERED	DIED	MORTALITY
Drainage through anterior abdominal wall	16	5	23·8
Drainage through loin below 12th rib ..	5	1	16·7
Drainage through pleural cavity ..	18	14	43·7

The above list confirms what one would expect to find, that when the presence of pus can be readily detected and efficient drainage established, then a relatively low mortality may be expected. The high mortality associated with drainage through the chest wall is partly accounted for by delay in searching for the pus, so that the general condition of the patient deteriorates before the abscess is discovered.

### CONCLUSIONS.

1. In this series of 78 cases of subphrenic abscesses, appendicitis was the cause in no fewer than 30, the commonest variety being right posterior intraperitoneal, of which there were 29 examples.

2. In cases of acute appendicitis in which the infection has spread beyond the appendix, expectant treatment reduces the incidence of subphrenic infection considerably.

3. In order to obviate pleural infection and obtain dependent drainage, a rib should be resected as low as possible, commonly the 10th. Posterior drainage should be carried out whenever possible; even when an anterior subphrenic space is involved, this can usually be accomplished.

4. The tendency of a right extraperitoneal abscess to extend forward between the layers of the falciform ligament was noticed in two cases, which suggests a possible route for drainage without involvement of the peritoneal or pleural cavities.

We desire to thank the surgical staff of the London Hospital for permission to include in this series cases under their supervision.

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**HÆMANGIOMA OF LEG.**

BY GEORGE BELL AND KEITH INGLIS, SYDNEY.

**CLINICAL REPORT (G. B.).**

THE patient, a female, age 17, was first admitted to Sydney Hospital on May 1, 1922.

When two years old she was kicked on the right leg, and soon afterwards a swelling was noticed. At the age of six years she was operated on by a surgeon, who is said to have restored the leg and foot to a normal size and shape. Radium has been applied to the tumour and caused no improvement. She has ceased to walk on the leg, which is now kept flexed on the thigh, with the foot in the position of talipes equinovarus. The right leg and thigh are wasted. There is a hard swelling in the leg, which extends from just above the ankle to the upper third of the leg posteriorly. The right thigh measured 37 cm. in circumference and the left 40.5 cm.; the right leg 35 cm., and the left 37.5 cm.



FIG. 436.—Showing the appearance of the limb in August, 1924, immediately before amputation.

In June, 1922, she was seen in consultation with Sir Herbert Maitland, who advised amputation at the knee-joint. The patient was unwilling to have the leg amputated then. On May 3, 1922, the Wassermann reaction was negative. During May, 1922, potassium iodide was administered, and also two doses of 0.3 gm. novarsenobenzol.

On May 29, 1922, under an anæsthetic, a piece of tissue was removed from the lateral portion of the leg at the junction of the middle and lower thirds. Dr. Keith Inglis examined this, and reported that the growth was a hæmangio-fibro-endothelioma.

The patient was again admitted to Sydney Hospital on July 28, 1924. She stated that the leg was usually not painful. Sometimes she felt sharp stabbing pains if she allowed it to hang down. The leg is gradually increasing in size, and the right is always warmer than the left. She had had radium and X rays applied.

On examination, the position of the leg and foot was much the same. The ankle could be flexed slightly, and there was also a slight range of inversion and eversion. The toes also could be moved through a fair range. The length of the right thigh was 37 cm., and that of the left 40.5 cm. The greatest circumference of the right thigh was 35.5 cm., and that of the left 44 cm. The lengths of the right and left legs were 33 cm. and 36 cm. respectively, and the greatest circumferences were 26.5 cm. and 29 cm. (Fig. 436). Two pigmented, slightly raised areas were present in the right leg, one over and one immediately below the medial malleolus. There was hyperidrosis of the right leg; the skin temperature of this leg was higher.

On Aug. 6, 1924, I performed a transecondylar amputation of the thigh. This healed by first intention, and the stump when seen during the last week of October, 1924, was an excellent one and showed no signs of recurrence of the growth.

Radiographic examinations were carried out and reports given as follows: (1) May 2, 1922: "Periostitis middle third of fibula; osteoarthritis of knee-joint." (2) July 15, 1924: "There is some periosteal roughening at the level of the tumour." (Fig. 437.)

#### PATHOLOGICAL REPORT (K. I.).

Within an hour after the limb was amputated an endeavour was made to perfuse it with Kaiserling's solution. All attempts to do this were unsuccessful, because the blood-vessels, even in the proximal end of the amputated limb, were abnormal, and appeared to merge insensibly in the angiomatous tumour. After removing a long slice of tissue from the posterior aspect of the leg it was found that a hæmangioma extended from the ankle almost to the knee (Fig. 438). The anatomical structures on the hind part of the leg were abnormal and ill defined, and the muscles were rendered inconspicuous by the extensive fibro-angiomatous tumour. It seemed as if the blood-spaces did not cause atrophy of muscle by pressure, but that the hæmangioma was actually in the substance of what would normally have been muscle. The large vascular sponge which permeated the muscles only slightly affected the adipose tissue and did not involve the skin (Fig. 438). It is probable that disuse and pressure were responsible for some of the muscle atrophy, but I am rather inclined to think that the almost complete absence of voluntary muscle on the posterior aspect of the leg was partly due to faulty development.

In order to study the tissues more thoroughly, a block 5 cm. long was cut out of the middle of the leg, where the changes in the bones



FIG. 437. — Showing the variation in density of the soft parts due to the tumour. The scattered deposits of calcium salts in the tumour can be detected. The effects on the bones are also evident. The maximum effect is just below the middle of the leg.

were most marked. This block of tissue included a portion of tibia and fibula and much of the soft parts around them. The skiagram of this segment showed the blurred outline of the bones and the bone spicules projecting into the soft parts. Horizontal sections of this segment revealed no definite posterior tibial artery—merely angiomatous tissue extending forward between the bones about as far as the normal situation of the interosseous membrane; but this membrane was not definitely recognized. In the front of the leg the muscles showed no abnormality, and the anterior tibial artery was evident.

Wollard,<sup>1</sup> in studying the development of the principal arterial stems in the forelimb of the pig, found that three stages could be distinguished: (1) The stage of the capillary net; (2) The stage characterized by large tubes showing island formation, coalescence, and a tendency to fuse—the retiform stage; and (3) The formation of a definite stem. I suggest that in our patient the small radicles forming the original net have in some areas failed to fuse, and, without fusing, have later become enlarged, dilated, and tortuous, to form a tumour in which both arterics and veins are represented. This would account for the absence of the posterior tibial artery by assuming, not that it had disappeared, but that it had never developed, and it would also explain the vascular sponge replacing the voluntary muscle.

The mode of extension is probably akin to that of nævi described by John Duncan.<sup>2</sup> I believe that what Duncan says of nævi holds true of angiomata in general. There are two modes by which a nævus may increase in bulk, he states, the intrinsic and the extrinsic. It may grow by the invasion of surrounding parts, or by pushing them aside through addition to its own internal bulk. Many a nævus runs a course which entirely precludes the idea of its increase being otherwise than by involving the surrounding tissues in its own mode of growth.

Microscopically the tumour in our patient is made up mainly of blood spaces of various sizes and shapes and filled with what appears to be normal blood. Some spaces contain granular material which is pink in preparations stained by hæmatoxylin and eosin. This is probably lymph. The presence of spaces containing lymph is deserving of comment. In many angiomata both blood spaces and lymph spaces are to be seen. It is true that in most angiomata one of the two predominates sometimes almost to the exclusion of the other, and in the present case it is the hæmangiomatous element which is by far the more conspicuous—so much so that by the naked eye the lymphangiomatous element cannot be detected.

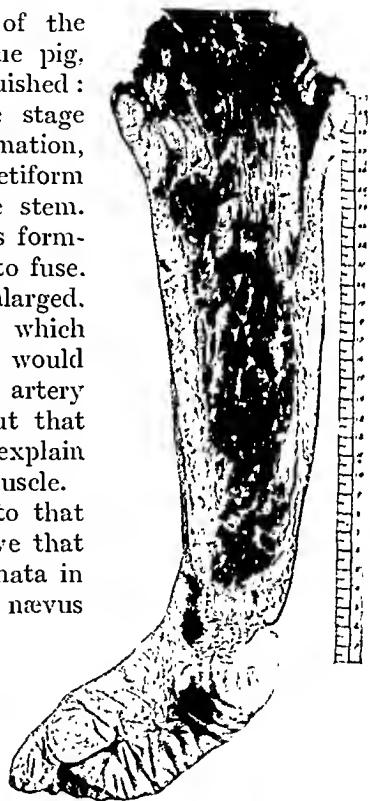


FIG. 438.—Showing the leg after removing a long thin piece of the soft parts from the dorsal aspect (centimetre scale).

In many situations the blood spaces are separated by very thin septa composed of flattened endothelial cells (*Fig. 439*), but elsewhere fibrous tissue in varying amount is also present between the blood spaces; indeed, in some portions of the tumour the blood spaces are so small and the fibrous tissue so great that the structure cannot be distinguished from that of vascular fibrous tissue such as is seen in inflammatory reactions. This dense fibrous tissue might be regarded as reactive and not actually part of the growth, as corresponding to the fibrous stroma of, say, a scirrhus cancer of the breast. There is, however, another possibility, and this I believe is the more likely. In granulation tissue endothelial cells may proliferate to form either vascular spaces or fibroblastic cells which, as the reaction progresses, become transformed into white fibrous tissue. The tumour under consideration is composed of the essential cellular elements of granulation tissue, and it is conceivable that here also two modes of development may be present, one into blood spaces, and the other through fibroblasts into white fibrous tissue.

Where the tumour meets the bone the changes are very interesting. I was anxious to see if, when this fibro-angiomatous growth of soft parts encroached on bone, the resulting changes would in any way simulate those seen in so-called osteitis fibrosa; but the resemblances are not as close as I expected to find them. The tumour is very fibrous near the surface of the bone, and projecting into this fibrous tissue from the bone are many spicules of various sizes. Fibroblastic and fibrous tissue can be seen filling the interstices in the thickened portions of the solid bone. In these situations the marrow within the bone is very vascular. In some of the interstices in the compact bone the connective tissue is very vascular. There is little doubt, I think, that the lesion began in the soft parts; then, encroaching on the bone, passed through it, possibly by way of natural foramina (the so-called Volkmann's canals), and extended to within the marrow cavity. The increased vascularity within the bone-marrow I regard as angiomatous and part of the lesion. Most of the spicules of bone were small (*Figs. 440, 441*); but in *Fig. 441* the whole of the bone depicted was separated from the main shaft. It seems as if a portion of the marrow was separated with it.

In the main growth are a few plasma-cells, eosinophil leucocytes, and fairly numerous small collections of lymphocytes. All of these may be the result of superadded inflammation; but I have found scattered accumulations of lymphocytes remarkably common in angiomatous growths, and wonder if lymphoid aggregations may sometimes be an essential part of the lesion.



FIG. 439.—Showing the structure of the angiomatous portions of the growth. ( $\times 60$ .)

Calcium salts have been deposited in the tumour as indicated in the skiagram (*Fig. 437*). The deposits are small and widely scattered. Most of them are situated in thrombi within the lumina of blood spaces; they correspond to phleboliths.

A case presenting many features in common with ours was observed by Bertram M. Bernheim.<sup>3</sup> This was a male whose left arm first bothered him when he was 16 years old. He then noticed a little red spot on the inner side of his wrist about 1.5 cm. in diameter. It gradually increased in size until he was 47 years of age, when the limb was amputated. The left arm



FIG. 440.—Showing the fibrous character of the growth near the bone (fibula), a bone spicule projecting into the soft parts, and connective tissue filling one of the interstices in the compact bone. ( $\times 60$ .)



FIG. 441.—The whole of the bone depicted in this figure was detached from the main shaft of the fibula. To the right a sharp spicule of bone can be seen projecting into the soft parts. Apparently a portion of marrow has been included in the detached portion of bone. The vascularity of the marrow is probably due to an extension of the angioma. ( $\times 30$ .)

was then twice the size of the right, and in the affected limb there were no blood-vessels; instead there were huge venous and arterial blood-sinuses that twisted and turned back on themselves. The muscles resembled a huge sea sponge, and arterial blood even spurted out of the marrow cavity.

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## THE SURGICAL TREATMENT OF CHRONIC BACILLARY DYSENTERY.

BY PHILIP MANSON-BAHR AND A. L. GREGG, LONDON.

CHRONIC bacillary dysentery, a very distressing and intractable disease, too often remains unrecognized in this country, and consequently does not receive the serious attention it deserves. In India, where it was first described by Crombie, it was recognized as a separate disease - the *Morbus bengalensis*; but this is now known not to be the case. Rather it is caused by, or is a sequel to, chronic ulceration of the mucous membrane of the large intestine by the bacilli of the Flexner-Y group. More rarely, as one of us has pointed out, it may be due to Shiga's bacillus, which is usually responsible for the more virulent and acute types of bacillary dysentery.

The chronic type of the disease is comparatively common in natives of India and other tropical countries, but is rarely seen amongst Europeans. During the last five years, however, a number of cases have occurred amongst ex-soldiers and pensioners from the Great War who originally contracted the infection on the Western Front or, more usually, in some of the Eastern theatres of war.

### PATHOLOGY.

The pathological appearances of the bowel naturally vary considerably in different stages of the disease. The less advanced lesions consist of shallow ulcerations which involve the mucous membrane alone (*Fig. 442*), and are found usually at the flexures of the large bowel, but may also occur in the lower ileum and extend into the rectum down to the anal margin. These ulcers are usually found on the free edges of the transverse folds of mucous membrane.

In contradistinction to the ulcerations of amœbic origin, they are serpiginous in outline, with undermined edges, and often intercommunicate with neighbouring lesions. Occasionally, at the base of the ulcers mucus-retention cysts (*Fig. 443*) form in the submucosa, in which the dysentery bacillus continues to exist in a virulent form, and it is these cases which usually become 'convalescent carriers' and materially assist in the spread of the infection. The isolation of the specific bacillus in these cases is usually possible only at autopsy; even then, it is by no means easy to obtain in any numbers. In order to do so successfully the bowel should be washed free from contents, and the base of the ulcer scraped and cauterized, cultures then being made from the granulation tissue by means of a platinum loop inserted under the margin of the ulcer.

In some cases this characteristic ulceration may, in time, involve the whole mucous surface, and, becoming secondarily infected by organisms from the faeces, may result in the deposition of exuberant granulation tissue. The

formation of granulation tissue is accompanied by fibrosis of the bowel wall, which, losing its elasticity, becomes thick and rigid. This process most



FIG. 442.—Shows the formation of a chronic bacillary ulcer affecting the mucous membrane only and not penetrating beneath the muscularis mucosæ. (Flexner-Y infection.)

usually affects the pelvic colon and sigmoid, which are the portions of the large intestine most severely involved.

The inflammation may extend to the peritoneal surface of the bowel, leading to the formation of granulations visible beneath the peritoneum.

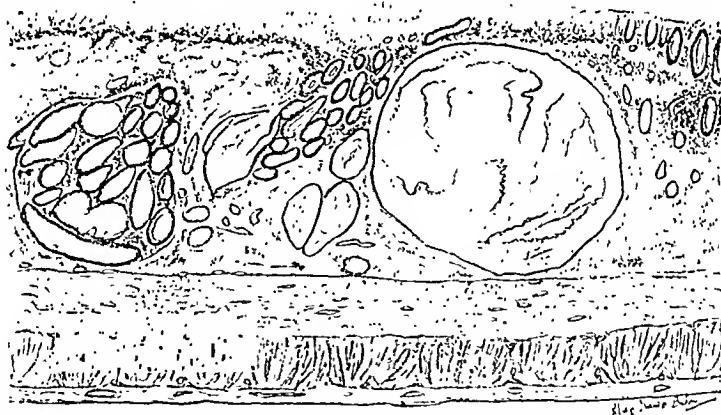


FIG. 443.—Shows the formation of submucous retention cysts, or adenomata, formed by the inclusion of portions of Lieberkühn's follicles in the scar tissue. (Shiga infection.)

Occasionally, too, large masses of granulation tissue may coalesce to form bleeding polypoid excrescences, and it is with this type of case that this paper is mostly concerned.

### SYMPTOMS AND COMPLICATIONS.

**Symptoms.**—The chronic stage of ulceration, or granular colitis, rarely, if ever, appears to be a sequel to an acute attack of bacillary dysentery. In the patients whose histories are quoted here, the chronic stage supervened

insidiously; they have suffered once, if not oftener, from a mild or subacute dysentery from which they made a rapid and apparently permanent recovery during their war service. Relapses of diarrhoea or subacute dysentery, with mucosanguinolent stools, recurred at definite intervals, or it might not be for a year or more after demobilization. Eventually a chronic, intractable diarrhoea supervened which, though temporarily yielding to rest and diet, finally produced weakness and emaciation, often with progressive anaemia, cachexia, and cardiac disturbances. The supervention of a chronic diarrhoea may therefore be regarded as the chief objective symptom of chronic bacillary dysentery.



FIG. 444.—Sigmoidoscopic appearance of acute bacillary dysentery. Shiga's bacillus isolated. (*Philip Manson-Bahr pinx.*)

In time, and as the deposition of granulation tissue progresses, the passage of diarrhoeic stools is replaced by evacuations consisting solely of blood and mucus, and this may be accompanied by acute dysenteric symptoms, such as acute abdominal pain and tenesmus. As may be gathered from the summary of our cases, eight to ten of these evacuations may be passed daily for weeks or months, till death from exhaustion takes place.

**Complications.**—In the latter stages of this distressing disease symptoms of general intoxication assert themselves, producing pains in the long bones and joints culminating in intra- and peri-articular effusions which may be acute and extremely painful.

Parotitis, a symptom not infrequently met with in acute bacillary dysentery, has also been noted in two cases.

Other stigmata of acute toxæmia may manifest themselves, such as bouts of pyrexia, complete anorexia, mental apathy, and stomatitis. A secondary, and maybe severe, anæmia is usually found.

## DIAGNOSIS.

The diagnosis of chronic bacillary dysentery usually presents considerable difficulties. On account of the failure attending the isolation of the specific organism, which has been already referred to, one has to rely, to a great extent, on negative evidence—the history of the patient, the continued absence of pathogenic amœbæ from the fæces, and the lack of response to anti-amœbic drugs.

The microscopic examination of the fæces may in itself afford a clue, for often masses of polymorphonuclear leucocytes derived from the bowel wall may be distinguished. The presence of these cells in diarrhœic fæces is there-



FIG. 445.—Sigmoidoscopic appearance of chronic bacillary dysentery showing glazed and granular appearance of mucous membrane due to formation of granulation tissue. (*Philip Manson-Bahr pinx.*)

fore very suggestive of chronic bacillary dysentery; in the amœbic disease these cells are usually entirely absent.

Little help can, as a rule, be derived from serological investigations. Occasionally, as in two cases in this series, the serum will be found to agglutinate Flexner's bacillus even in high dilutions, i.e., 1-100 or more, and this test may be of suggestive value. More usually, however, it affords little confirmatory evidence.

On the other hand, sigmoidoscopic examination of the bowel is most valuable and is usually diagnostic. From the fact, already stated, that the most marked lesions occur in the lower part of the large bowel, and especially in the rectum, they are usually clearly visible when the instrument has been introduced a few inches (*Figs. 444, 445*).

The distinctive features of the bowel are sufficiently apparent. The bowel itself is rigid, indurated, and inelastic, due to long-standing inflammation, a

condition which gives rise to a considerable degree of pain on the introduction of the instrument; this in itself is of considerable diagnostic value, serving to distinguish it from amœbic ulceration, which we have found to be almost invariably painless. The roughened and granular surface from which blood exudes wherever touched by the instrument is also sufficiently distinctive.

The main feature of this bowel is the granulation tissue itself; naturally the appearance varies in different cases, and in some it may be actually polypoid. Fibrinosis of portions of the bowel resulting in localized strictures has also been noted. The patulous condition of the anus and the atrophic appearance of the skin surrounding the anal margin, as well as the wasting of the gluteal and perineal muscles, afford a considerable amount of additional evidence in forming a diagnosis. Redundant skin at the anal margin, and the formation of external piles, which so often accompany chronic amœbic dysentery, are seldom, if ever, seen in the chronic bacillary disease.

### MEDICAL TREATMENT.

From a brief consideration of the pathological aspects of this serious condition, it is hardly a matter of surprise that drug treatment is of little, if any, avail.

Such drugs as bismuth, salol, and bolus alba merely serve to mask the symptoms temporarily, but cannot in any way mitigate the serious condition of the large bowel. The same may be said of vaccines. The specific organisms, the dysentery bacilli, play little, if any, part in the terminal stages of bacillary dysentery, so that the destruction of the mucosa and its replacement by granulation tissue have to be regarded as the aftermath of their activities. Under these circumstances we have come to regard vaccines of dysentery bacilli not only as entirely ineffective, but as actually contra-indicated. The same may be said of mixed vaccines composed of various species of the intestinal flora. The problem really becomes a mechanical one; one's efforts should therefore be directed towards diverting the fecal stream as much as possible from the large gut, and to putting this viscus in a state of almost complete rest, so that healing may take place. Apparently regeneration of the epithelium rapidly takes place when the source of irritation is removed (*Fig. 446*).

The lines of treatment may be divided into three headings: dietetic measures, intestinal lavage, and operative interference.

**Dietetic Measures.**—In contradistinction to the usual practice adopted in dieting chronic amœbic cases, we are of the opinion that in the chronic bacillary disease it is necessary to supply the patient with an adequate supply of nutritious nitrogenous food. Oils and fats are particularly useful in so far as in excess they tend to lubricate the raw surface of the bowel and thus promote healing. The main aim should be to stimulate the recuperative powers of the body so as to aid the regeneration of the intestinal mucosa. This principle is also advocated by Lockhart-Mummery and others engaged in the treatment of ulcerative colitis and other conditions of the colon. If the patient has zest for food, the diet should not be restricted to milk and fluids, but he should be permitted eggs, fish, milk puddings, minced mutton,

jellies, and plenty of butter and bananas. Apparently it is not the nature of the faecal residue which irritates the large gut, but the fact that excrementitious matter is passing through the lumen at all.

**Lavage.**—In the milder stages of this condition, that is, in cases characterized by diarrhoea without grave constitutional disturbance, treatment by lavage per rectum sometimes appears to be followed by good results. In many cases, however, the effect is merely temporary, and after a brief period of quiescence, the process becomes progressive once more.

Apparently the substances employed in the lavage, either as means of disinfecting the bowel surface or of stimulating the mucous membrane to repair, are not so important as the amount of fluid used to flush out the bowel. The therapeutic agents we employ at the Hospital for Tropical Diseases for this purpose are sodium bicarbonate in a 4 per cent solution, hypertonic saline, and eusol. The latter has definite antiseptic properties,

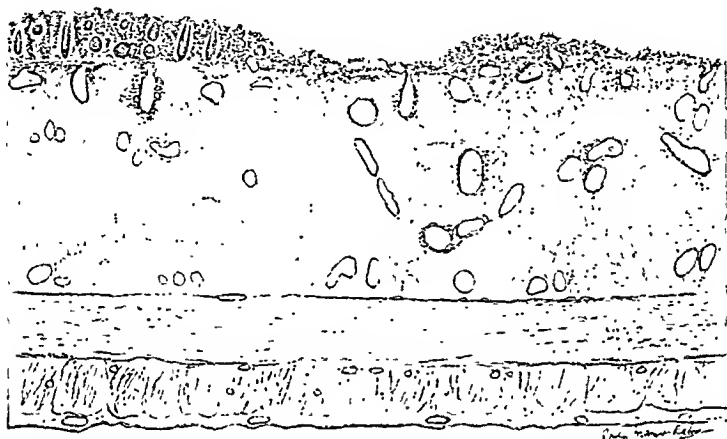


FIG. 446.—Active formation of granulation tissue on the mucous surface is taking place. The regeneration of the few remaining Lieberkühn's follicles can be seen on the left-hand side of the section, representing the process of healing.

and is therefore useful, but in addition it acts as a stimulant for the repair of the bowel. It is, however, as a general rule, most irritating, and it is necessary to dilute it very considerably in order that it may be tolerated by the patient. A 25 per cent solution of eusol is in routine use, but it may occasionally be used less highly diluted, though it produces a considerable amount of intestinal pain and, exceptionally, a toxic nephritis with hæmaturia. The other two substances tend to soothe intestinal irritation.

As regards the method of administration, the bowel should be washed free from intestinal contents by means of a tube and funnel and hot water. When the water is returned reasonably clear per anum, the solution should be run slowly into the rectum by means of a rubber tube and funnel. The tube itself should be a stout one provided with a round terminal opening, and it should not be inserted high up into the rectum, for in this situation it tends to become kinked and injure the mucosa. Not less than two pints of the solution should, if possible, be used to flush out the large gut, and the

fluid should be run in slowly, the patient in the meantime lying on his right side, and endeavouring to retain the fluid as long as possible. The time he is capable of retaining it varies considerably in individual cases from five minutes to half an hour. We are of the opinion that methods to ensure the flow of fluid through the gut, such as tipping up the end of the bed on blocks, or of inverting the patient himself, do not make any material difference.

We have recently come to regard intestinal lavage with silver salts, a method formerly in vogue, as being actually deleterious. Silver nitrate in a  $\frac{1}{4}$  per cent solution is extremely irritating to the bowel in this condition, while protargol and argyrol, which are usually employed in about 4 per cent strength, are far too expensive for routine use.

On the whole the results of lavage are disappointing, though a large number of cases have been treated; in the worst, resort has eventually had to be made to operative interference. We have notes of eleven cases during the last two years treated by lavage on alternate days, in all of which sigmoidoscopic appearances showed slight formation of granulation tissue, and in which the regeneration of the mucous membrane could be observed through the sigmoidoscope to take place. The average stay in hospital was about four weeks, and each patient received about twelve treatments or more. Intestinal lavage is particularly exhausting to the patient, and no object appears to be gained by washing out the bowel more frequently than is outlined above.

One case may be quoted in support of this method :-

*Case 1.*—The patient contracted dysentery in France in 1917, and suffered almost continually from diarrhœa and dysenteric symptoms for six years before the treatment was applied. He had lost some three stone in body weight, and had become feeble and cachectic. Treatment by means of enol and saline lavages was continued, with interruptions, over a period of nine months. On discharge from hospital the patient had regained his original weight and was passing two formed motions a day. Sigmoidoscopic examination showed that the bowel had regained its normal appearance, and the patient has been able to resume his occupation as a chauffeur.

### SURGICAL TREATMENT.

**Indications for Operation.**—As a result of our experience we may state that the indications for operation should be based upon the following: signs of toxic absorption, marked intestinal hæmorrhage, sigmoidoscopic appearances indicating grave destruction of the mucosa, the formation of bleeding polypi, and the failure of properly conducted lavage. We are further of the opinion that it is not justifiable to wait till the patient is almost *in extremis* before instituting operative measures. Should the patient not respond to lavage within a reasonable time, should he continue to lose weight, the indications are that persistence in these measures will not meet with any greater degree of success, so that recourse must be had to operation. Inasmuch as dental treatment is a form of surgery, it must be accepted that no treatment of dysentery can be entirely successful if oral hygiene be neglected.

Two methods, represented by four operations, may be considered:

- (1) Through-and-through lavage obtained by appendicostomy or valvular

cæcostomy; (2) Faecal drainage obtained by an open cæcostomy or ileostomy. Colostomy, apart from the rare event of stricture, has no place in the treatment of dysentery: if the disease is grave enough to warrant operation, almost the whole large gut, and certainly the cæcum, will be ulcerated; there can be no rationale for leaving a portion of diseased gut, such as the cæcum, proximal to the opening.

**Appendicostomy and Valvular Cæcostomy.**—These may be considered together, being identical in effect. Valvular cæcostomy is more simple to perform where a minimum of manipulation is important, but an appendicostomy removes a latent source of infection with an almost certainly diseased appendix. For this sufficient reason we prefer this method. Either operation can usually be performed under local anæsthesia; it causes little discomfort, and the wound, when permitted, will heal spontaneously. These advantages are outstanding, and the results frequently most gratifying; hence the well-deserved popularity of this method. With a small catheter in the cæcum, complete flushing of the large gut can be assured, or, by using the continuous drip, many ounces of fluid can be slowly passed into the bowel, from which it is readily absorbed by the dehydrated system. Such procedure, however, can only ensure success when adopted early. With other writers, we feel the need to emphasize this point—alas, too often overlooked.

Where the patient's recuperative powers have been undermined by long-continued toxic absorption, the success of appendicostomy will be problematical; indeed, it has been our experience that long-standing cases presenting the characteristic clinical picture of marked toxic absorption, together with the constant passage per rectum of blood, mucus, and pus, do not always obtain benefit from a small opening; complete drainage being required. The following history is illustrative:—

*Case 2.*—L. W., age 29, ex-soldier, contracted diarrhœa in France in 1916: intermittent relapses occurred till May, 1923, when a severe attack of hæmorrhagic diarrhœa caused his admission to the Hospital for Tropical Diseases. Dietetic and lavage treatment was instituted. Bilateral parotitis developed in June, and subsided without suppuration. By October, 1923, symptoms had improved and the patient was discharged. Re-admitted November, 1923, passing much blood, mucus, and pus per rectum.

Sigmoidoscopy revealed a congested granular and bleeding mucosa. Local 2 per cent solution of silver nitrate was applied to granulations, with no improvement. On Jan. 12, 1924, appendicostomy: still no improvement: patient now becoming emaciated and very toxic. On Feb. 13, 1924, open cæcostomy was done: it was followed at once by slow but steady improvement, patient gaining in weight and general health. At the time of writing, blood and pus are still passed per rectum, but all toxæmia has vanished. The cæcostomy has not yet been closed, but patient is able to get about, is in good health, and has increased three stone in weight.

For the purpose of through-and-through lavage we have found nothing more soothing and generally satisfactory than hypertonic saline to which occasional additions of eusol (5 oz. to the pint) are made. Lavage was usually done once daily.

**Open Cæcostomy.**—This also is a simple operation and can be done under local anæsthesia, but it should be noted that the gut is greatly inflamed, and usually a subacute peritonitis is present. The peritoneal nerve-endings are therefore in an irritable condition, so that even the most gentle



handling is liable to cause a dull aching pain. Two preliminary injections of morphia,  $\frac{1}{2}$  gr., given ninety and thirty minutes respectively prior to the operation, are therefore very desirable. Gas-and-oxygen anaesthesia may be preferred. Our practice has been to make a muscle-splitting incision over the caecum, draw forth the appendix and as much of the caecum as may be judged necessary, and then shut off the peritoneal cavity by suturing the cut edges to the caecal wall. A Paul's, or  $\frac{1}{2}$ -in. rubber, tube is next inserted into the caecum through an elliptical incision which includes the base of the appendix, this organ being thereby removed. Such practice renders remote a chance infection of the peritoneum and obviates leaving a dangerous appendix. We make a point of not suturing the skin too closely around the tube; hence the wound infection which follows is mild in nature, free escape of any exudate being permitted.

One unexpected source of difficulty will surprise the surgeon unfamiliar with dysenteric ulceration. This arises from the marked degree of oedema and thickening present in the caecal wall, which greatly hinders the introduction of the Paul's tube. An allowance of caecum beyond the peritoneal sutures which would appear amply sufficient for the introduction of the tube may prove totally inadequate when an incision has been carried through the thickness of the gut—only an astonishingly small opening being obtained. Full provision for this must therefore be made before the original peritoneal sutures are inserted. On the other hand, all possible caecal wall must be left in the abdomen; by so much is the eventual closure facilitated. It is because of this difficulty that a delay is not made before opening the caecum.

Nursing difficulties commence in a week or less with the loosening of the tube, as do the patient's unpleasant experiences. The liquid and digestive faecal material from the opening is both offensive and irritating; the former can be met by the free use of sanitas or eusol and eau de Cologne; the latter by painting the skin around the wound with ambrine at each dressing. A colostomy belt should be fitted as soon as possible.

As regards attaining the ideal of complete drainage of the faeces, it is well known that this procedure fails; peristalsis still drives a small amount of the material along the colon.

To combat this tendency one of us (A. L. G.) devised the tampon-tube illustrated in *Figs. 447, 448*. It is both simple and efficacious when used as follows:—

The tampon-tube is simply a soft rubber bulb which is fastened round an ordinary rectal tube and can be inflated at will. It is made by Messrs. Allen & Hanburys. With the bulb B (*Fig. 448*) deflated, the tube A is passed through the caecostomy until the bulb has just passed into the ascending colon. Gentle inflation from a syringe of a known quantity of air through tube C then prevents the bulb from returning through the wound. The glass rod D is now pushed through tube A at skin level as an



FIG. 447. —Tampon-tube.

anchor to prevent any advance of the bulb from peristaltic pressure. The lumen of the ascending colon is thereby blocked so that the onward passage of faecal material becomes impossible. At the same time the central tube A permits of colonic lavage when required, reflux being prevented by a simple clip, E. By this means all the comforts of appendicostomy lavage are gained, while drainage is assured.

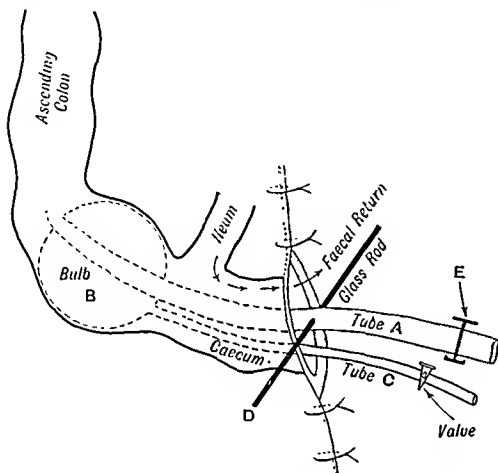


FIG. 448.—Diagram to show tampon-tube *in situ*.

was performed with local anaesthesia, and Paul's tube was inserted. Toxaemia forthwith decreased and the condition improved, though rectal haemorrhage continued for some weeks. Using the tampon-tube, through-and-through lavage was continued, and gradually the patient's appetite returned, weight was gained, and the haemorrhage ceased. A colotomy belt was then worn for some time, and by June 30 the patient was so much better that the caecostomy was closed. Uninterrupted convalescence occurred, and he returned home at the end of July, 1921. He was last heard of in December, 1922, up to when he had maintained excellent health and had no bowel trouble other than occasional slight discomfort in the abdomen.

Once an open caecostomy is established, the patient may be expected to improve from even the most hopeless condition. Such improvement, unfortunately, does not decrease the surgeon's anxieties, for he has yet to face the difficult problem of when to close the wound. Manifestly a permanent right-sided anus is objectionable, but prior to any operation the patient should be warned that such may be inevitable. We accept three criteria as signifying the possibility of closure:—

1. *The general clinical condition*: The patient must have gained in weight and general health, no evidence of active toxic absorption still being present.
2. *The absence of ulceration in the colon*: To determine this, *both ends of the colon are examined*; the distal end by an ordinary sigmoidoscopic examination, the proximal end by means of a cystoscope passed through the caecostomy. If the bowel is flushed through from the anus and a clear gentle stream is kept flowing, it is surprising what an instructive view can be obtained of the whole ascending colon.
3. *Blood should have been absent from the irrigation result for some time, and no recurrence or pain should occur when the faecal material is encouraged to pass through the colon again.* This can be done by applying a firm flat dressing

to the cæcostomy after the tampon-tube is removed, faecal exit through the wound being thus hindered.

Not until these conditions are satisfied is it safe to close the wound, nor will the patient ever be exempt from either immediate risks or a remote relapse. For these reasons one hesitates to close the opening, and, as others have noted, such hesitation may well be justified. None the less the patient will concur that it is well worth considerable risk to be relieved of a right-sided abscess, and that confidence in the patient's recovery may be justified is illustrated by the following history:—

*Case 4.*—A. V. C., age 27, ex-soldier, contracted diarrhoea in France in 1917, with passage of blood and mucus. There were occasional recurrences up to June, 1920, when he was admitted to the Hospital for Tropical Diseases. He was then very anæmic and passing much blood and mucus. Tachycardia, abdominal pain, and irregular pyrexia were marked: the condition was very suggestive of tuberculous, and became critical. Treated by diet, salines, lavage—all useless. On July 17 one of us (A. L. G.) was asked to perform open cæcostomy. Following this, through-and-through lavage kept the colon clear. The patient's general condition and local symptoms slowly improved, so that on Oct 6 the cæcostomy opening was closed. Both operations were done under general anaesthesia. Uninterrupted convalescence followed and patient was discharged on Nov. 1, 1920. He resumed his trade as blacksmith some weeks later, and still follows it without any disability or recurrence of dysentery. In October, 1925, he reported that he was perfectly fit and well and enjoying life.

**Ileostomy.**—This has been advocated to meet the drawback of cæcostomy already mentioned, viz., the passage of some faecal material through the colon.

We have carried out this method: it must of necessity relieve the large bowel completely; it is simple to perform, and no more dangerous to close than a cæcostomy. Against ileostomy, however, we urge the loss of an important sphincter and portion of the digestive tract. The use of the tampon-tube permits the advantages, hitherto obtained only by an ileostomy, of complete faecal drainage. Further, the possibility of being able at will to direct the faeces through the colon when estimating the time for closure is an immense advantage which is ruthlessly sacrificed by ileostomy.

There remains, then, but one definite indication for ileostomy, namely, an extension of the inflammatory process into the lower portion of the ileum. Should such grave instances arise—a rare occurrence in chronic cases—we prefer to make the bowel opening a short distance proximal to the ileo-cæcal sphincter and to the inflamed gut. The following patient illustrates such a condition:—

*Case 5.*—W. S., age 45, ex-soldier. Served in France 1916–19. Diarrhoea with blood began in 1920 and lasted three months. It recurred in September, 1923, and continued up to Feb. 18, 1924, when he was admitted to the Hospital for Tropical Diseases. Average of six stools daily, with much blood, mucus, and pus.

Sigmoidoscopy: examination painful; granular bleeding bowel, which was narrowed and fibrotic, elasticity being lost. Lavage treatment failing, on March 7, 1924, a loop of the ileum was brought out of the abdomen, as the inflammatory condition of the bowel was seen to extend about 8 in. into the ileum. Three days later the bowel was divided and a catheter introduced into the distal end. Through-and-through lavage was maintained. Stronger solutions of eusol were tried, but an

acute nephritis followed, fortunately only transitory. Apart from this, steady gain in health ensued, although the passage of blood and pus per rectum continued.

The patient was discharged on June 14, 1924, and reports from time to time. His general health and condition are now good, but the passage of blood and pus persists and contra-indicates restoration of the alimentary tract.

It has been pointed out that, after some months' disuse, a bowel badly ulcerated originally, may become so stenosed that any attempt to close the artificial anus is doomed to failure from partial obstruction. That such a condition may occur was demonstrated at the autopsy on one patient whose death was in the nature of a calamity. Possibly this patient had had both amœbic and bacillary dysentery, but we are sceptical as to the finding of the dysentery amœba as reported.

*Case 6.*—H. H., age 25, ex-soldier, served in Salonika, where in 1917 he contracted dysentery, and *Entamœba histolytica* was reported to have been found. He



FIG. 449.—*Case 6.* Sigmoidoscopic appearance of chronic bacillary dysentery showing partial fibrosis of the bowel wall and formation of ridges of granulation tissue. (*Philip Manson-Bahr pinx.*)

was treated with emetine, but in spite of this had continual relapses for six months. These gradually cleared up with bowel lavage, but in 1918 and again in 1919 he had severe relapses. On Nov. 8, 1921, he was admitted to the Hospital for Tropical Diseases, very ill with severe dysentery symptoms. Patient was very toxic and very weak from loss of blood, his condition being critical, so that on Nov. 10, one of us (A. L. G.) was asked to do cæcostomy. A portion of the cæcum was sectioned, and showed well the œdematous musculature, small-celled infiltration, and hæmorrhage at the base of the necrosed mucosa. The tampon-tube was used, and the patient slowly but steadily improved. Sigmoidoscopy in January, 1922, showed the bowel to be very granular and fibrotic (Fig. 449), and sphincteric control was absent. From being in a state of extreme emaciation and anæmia, patient became well covered and reasonably robust. Progress was interrupted by a thrombosis of the left femoral

vein, but this subsided without ill effects. Throughout, the patient complained of tenderness over the sigmoid colon.

On May 23, 1923, the case-sheet records: "Motions formed; general condition greatly improved; wound getting smaller." The cæcostomy opening became so stenosed that it ceased to function properly, and pain recurred in the abdomen. On July 7 a finger was introduced into the cæcum to dilate the opening slightly. Next day the patient was much more comfortable, but on July 9 all the signs of acute septicæmia suddenly developed, and death occurred on July 11.

Autopsy findings were those of acute septicæmia, and only *B. proteus* was isolated. In contrast to the previous emaciation the body was excellently nourished, and the colon was almost completely healed. There was in the sigmoid a markedly stenosed portion about 1½ in. wide—evidently the site of a severely ulcerated area and of the patient's tender spot.

It will be seen from the above that only a small portion of the bowel was stenosed; we believe that this is what may usually be expected in cases

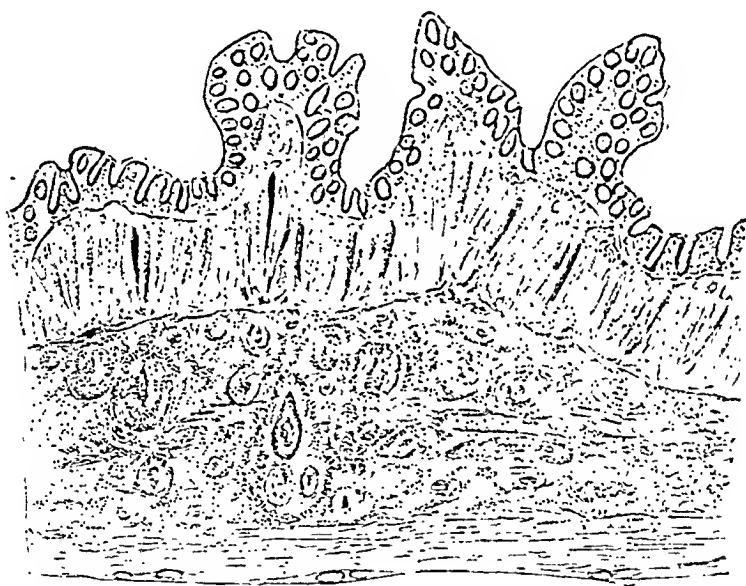


FIG. 450.—Tuberculosis of longitudinal muscular coat of the large intestine secondary to bacillary dysentery.

where stenosis occurs. We therefore prefer to close a cæcostomy working first from within the abdomen through a paramedian or mid-line incision, which permits of a careful preliminary inspection of the whole large bowel. Any unduly constricted portion can then be detected, safely removed, and an anastomosis carried out, closure of the cæcostomy being, of course, postponed.

There is one danger of which we were previously unaware. We have come to regard the coexistence of tuberculous infection of the large bowel engrafted on to chronic bacillary dysentery as a serious danger. It is possible that the tubercle bacillus finds a congenial nidus in the previously existing granulation tissue, or becomes spurred into activity by the general devitalization of the mucous surface, and in some cases this has clearly happened, the

tuberculous process appearing to extend chiefly in the circular muscular coat of the colon (*Fig. 450*). In three cases in the period covered by this work, we have been able to demonstrate tubercle bacilli in scrapings made from the bowel wall through the sigmoidoscope.

We are of the opinion that the results obtained in this limited number of cases are striking, and justify the view that operative measures are strongly indicated as being the only rational method in this singularly chronic and intractable disease.

### SUMMARY.

1. Bacillary dysentery of a mild type may result in a progressive and chronic ulceration of the large bowel in which the specific dysentery bacillus plays but the initial rôle.

2. The resulting condition is usually not amenable to medical treatment, and life may be preserved only by surgical intervention.

3. Indications for operation lie in the persistence of toxæmia and loss of weight in spite of colonic lavage.

4. Appendicostomy may be performed as a measure of relief, but cæcostomy may be required.

5. Cæcostomy with the use of the tampon-tube supplants ileostomy except in cases in which the small bowel is involved.

6. The question of closure is a matter for careful consideration in each case; temporary closure with dressings assists the operator in making his decision.

7. Stenosis of the bowel to any serious extent is rare, and when it does occur is likely to be localized, so that excision may be practicable.

8. Tuberculous infection may be superimposed upon a chronic ulcerated dysenteric bowel.

Our best thanks are due to Dr. W. F. Whaley, lately House Physician to the Hospital for Tropical Diseases, for his help in collecting the information contained in this paper; and to Dr. G. C. Low for his kind permission to make reference to cases under his care.

## THE CLOSURE OF CONGENITAL CLEFTS OF THE HARD PALATE.

BY ANDREW CAMPBELL, JOHANNESBURG.

ON considering the various operative measures in use at the present day for the closure of congenital clefts of the hard palate, there are a few points in the technique which strike the operator as being somewhat unsound and capable of improvement. Each cleft-palate defect is a problem presenting difficulties which have to be overcome by the most suitable methods.

When a unilateral cleft of the hard palate associated with a high palatal arch is seen, the surgeon does not experience much difficulty in making up his mind, because ample flaps are available and also a choice of operations. On the other hand, if there are bilateral clefts with scanty tissue available for flaps, then he is not in such a happy position. If the flaps have to be cut large, there is left a correspondingly large raw surface which cannot be avoided. This raw surface left in the mouth after operation is a source of anxiety to the surgeon, and is probably the most frequent cause of operative failure. The introduction of stitches is a tedious part of the procedure, and it requires considerable patience and skill before the suture line is considered to be satisfactory. Often a few of the individual stitches tear out after completion of the operation, and no matter what care is taken, there is bruising and tearing of the tissues in the most vital part—the suture line. The larger the number of stitches introduced, the greater is the traumatism. A certain number of surgeons must have concluded that the methods now in use are not satisfactory, otherwise they would not recommend the wearing of an obturator after the closure of the cleft in the soft palate. An obturator must be a nuisance to everybody concerned; it is quite impracticable in growing children owing to the necessity of changing it frequently; there is difficulty in fitting; it is frequently not worn; and it harbours débris, etc., rendering the nasal passages more prone to infection. It may be dislodged and become impacted in the pharynx or elsewhere.

It is not the intention of the writer to belittle the classical operations which have been so successfully performed, but he wishes to point out certain disadvantages, which may be summarized as follows: (1) A low palatal arch and a wide cleft are difficult to deal with by any operation; (2) Bilateral clefts require large flaps, and these leave large raw surfaces in the mouth; (3) The large number of stitches introduced in most operations and the consequent injury to tissues devitalize the line of union; (4) The time occupied in operating in all but simple cases is unduly long; there is considerable hæmorrhage and shock, while death follows in some cases.

The following description refers to an operation which is practised by the writer and overcomes those disadvantages which have been detailed.

The operation evolved itself in the case of an adult female with a complete unilateral hare-lip and cleft palate. No one had made an attempt to operate on this patient, and she presented herself at the out-patient department for relief of a deformity which must have been a source of embarrassment to herself and to others. The gap was wide and the septum was deflected, as is usual, to the same side as the gap. It seemed obvious to a nasal surgeon that it would be easy to employ the septal tissue in the closure of the gap. This was accordingly done with success.

The original intention was to turn over the lower part of the septum complete with cartilage and attach it to the lateral margin of the gap, but the cartilage became detached and consequently only the two opposed mucoperichondrial flaps were utilized in the closure. It formed a serviceable palate, and after the hare-lip operation the patient left the hospital satisfied with the result. She visited us recently after a lapse of nine months, and the condition of the hard palate is still satisfactory.

It is to be noted that no raw surface except the line of suture was exposed in the mouth, and the loss of the septum was of little consequence to the patient. There are many individuals who have lost a large part of their nasal septa through no fault of their own and are none the worse for it. This particular patient is quite comfortable, with a serviceable hard palate, in spite of the absence of perhaps a quarter of her nasal septum.

This case led the writer to think more of the nasal septum as a tissue eminently suitable for the purpose of filling defects of the hard palate. It is within easy reach, there is enough septal tissue to fill the largest imaginable gap, it is highly vascular, and it heals well under very adverse conditions; while a loss of part of it is not detrimental to the patient.

### TECHNIQUE OF OPERATION.

The technique of the operation as performed at present is as follows, and the description is applicable alike for a unilateral complete or incomplete cleft of the hard palate. A bilateral cleft may, for purposes of this operation, be looked upon as a unilateral cleft on both sides, with the septum conveniently free in the middle line for the removal of adequate flaps.

The first step consists in the formation of the *palatal flap*, and involves the reflection of a flap from the buccal surface of the palate on the same side as the cleft, with its base on the lateral margin of the cleft. The width of this flap is approximately a little larger than the width of the cleft—that is to say, if the cleft were uniformly 1 cm. wide, the flap would be 1.2 cm. wide. The incision is made parallel to the cleft margin and goes down to bone. It extends as far as the posterior border of the hard palate, and the ends of the incision are then joined to the margin of the cleft. The mucoperiosteum is reflected medially as far as the margin of the gap in the bone, and the hinged flap thus formed is turned upwards so that it comes to lie with its medial edge in apposition to the lower edge of the septum and with its raw surface looking downwards into the mouth (*Figs. 451, 452*).

The second step consists in the formation of the *nasal flap*, and this is carried out by measuring the distance between the lower border of the septum



and the unreflected or lateral edge of the palatal incision. If we suppose this to be 1.2 cm. posteriorly at the junction of the soft and hard palates, then we place a mark on the nasal septum about 2.5 cm. vertically above its lower border. If the gap narrows anteriorly to 0.5 cm., then again we mark a point above on the septum 1.2 cm. from the lower border. A line of incision is thus outlined on the mucous membrane of the nasal septum. With a rectangular knife a horizontal incision is made from behind forwards along this line, cutting through the mucoperichondrium as far as, but not into, the cartilage of the septum. The anterior and posterior ends of this incision are now joined to the lower border of the septum. With an elevator the mucoperichondrium is turned down so that it hangs as a curtain in the mouth. This nasal flap has its base at the medial margin of the cleft, its raw surface looking into the nose and its lateral edge in approximation with the lateral edge of the palatal incision (*Figs. 451, 452*).



FIG. 451.

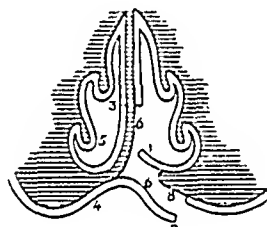


FIG. 452.

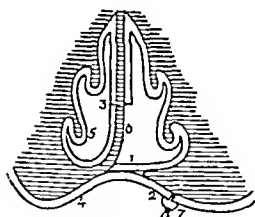


FIG. 453.

FIGS. 451, 452, 453.—Operation for cleft palate. 1, Palatal flap; 2, Septal flap; 3, Nasal septum; 4, Palate; 5, Inferior turbinate; 6, Septal raw surface; 7, Suture line; 8, Palatal raw surface.

The third step consists of the introduction of one or two sutures uniting the upper and lower flaps at the base of the septal flap. This, however, is not always necessary; but the lateral edge of the nasal flap is sutured to the line of the palatal incision with three or four stitches. This completes the operation, and after a little practice it does not take more than twenty minutes to perform it as described.

On reconsidering the procedure, we find that nasal raw surface has been applied to palatal raw surface and the line of union is no longer a line but a broad plane of union (*Fig. 453*). There is no raw surface in the mouth or in the floor of the nose. The cleft is covered by two flaps, one above the other, both of which contain periosteum. A small perforation made during the elevation of the flaps is therefore not a very serious accident. A palate composed of two flaps and with an absence of raw surfaces above and below is

obviously in a healthier condition than one where two palatal flaps have been united end to end, or where there is only one flap with its raw surface looking towards the mouth. Stitching has been reduced to a minimum, and it does not matter whether the cleft is wide or narrow, whether the palate is high or low. The only raw surface remains on the side of the nasal septum from which the septal flap has been cut. We have thought of grafting this surface with epithelium, but so far have not attempted it. It should be easy to retain such a graft with a little gauze packing, provided the graft was applied prior to the suturing of the flaps. If grafting is considered necessary, the method used for grafting the cavity after the radical mastoid operation is recommended.<sup>1</sup> However, so far we have found that the raw surface on the septum is of little consequence. There is oozing for a few hours and a little serous discharge for a few days longer. There is no tendency to sloughing of the septal cartilage, and this is not surprising because of the excellent blood-supply from the opposite side; also the nose is more or less sterile, as nothing but fresh air passes over the raw surface. If one deals with a bilateral cleft, the same operation is done on the other side, but it is wise to wait until the raw surface on the septum is entirely healed—a period of from four to six weeks (*Fig. 454*).

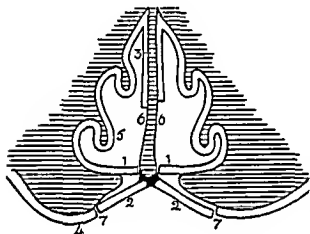


FIG. 454.—Operation for double cleft. 1, Palatal flap; 2, Septal flap; 3, Nasal septum; 4, Palato; 5, Inferior turbinal; 6, Septal raw surface; 7, Suture line.

Only a few minor points remain to be commented upon. In children the amount of septal tissue available is always plentiful and well nourished if the patient is in a suitable condition for operation. In adults we have not as yet had enough experience to make any definite statement; sufficient tissue must be available, but it may be very thin. The presence of septal tissue in the mouth gave us some uneasiness to begin with, but all the sensory branches of the fifth nerve and branches of the olfactory nerve supplying the nasal flap must have been severed by the incision.

Therefore there is no tendency to sneezing or other unpleasant sensations on contact of food with the new hard palate. It is more convenient to close the cleft in the soft palate first by the usual methods before attempting the closure of the hard palate and alveolus. It is preferable to leave the hare-lip, if any, to the last, as it provides more room for working comfortably in the nose. Cases are met with where the hare-lip has been closed by others, and in one recent case with which we dealt the cleft had been complete originally, but the alveolar part had been united by operation or had obliterated itself after the hare-lip operation. This closure of the hare-lip did not present any difficulty in forming the septal flap.

A word of warning is necessary in relation to the separation of the nasal flap. The mucoperichondrium of the nose separates easily, but it is also apt to be easily perforated, and when this occurs a small perforation is liable to gape widely. The palatal flap, however, will fit over the perforation. A knowledge of the anatomical relations of the septal cartilage and perichondrium, with the vomer and its periosteum, will reduce the possibility of perforation to a minimum.

The operation is by no means one restricted to the nasal surgeon. The bleeding is at no time troublesome, especially if a little weak cocaine is packed against the septum for a few minutes after the patient is under the anæsthetic. The use of a suction apparatus to free the pharynx of any blood and secretion which may collect is strongly recommended. Only one assistant is necessary, who has little to do except prepare the stitches and have the swabs ready. The instruments required are very few: a suitable rectangular knife for the septal flap, a scalpel for the palate, one small elevator, one pair of toothed forceps, and a few small half- and fully-curved needles and needle holder (Lane), with fishing gut or even catgut.

The after-treatment is almost negligible, except that the operator's attention is focused on keeping the nose clean by means of a mild oily antiseptic spray. The stitches may be removed in from four to ten days. Hæmorrhage is more to be feared from the nose than from the palate, but so far it has not been found necessary even to pack the nose.

The method has been sufficiently successful in our hands to recommend its trial by other surgeons, and it is hoped that they may record their results of a departure from the classical operations at present in general use.

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#### REFERENCE.

- <sup>1</sup> *Jour. Laryngol. and Otol.*, 1923, Sept., 471.

## SIMPLE TUMOURS OF THE LARGE INTESTINE AND THEIR RELATION TO CANCER.

BY CUTHBERT DUKES, LONDON.

THAT simple tumours of the mucous membrane of the large intestine are often followed by a cancer of the bowel wall is well known to pathologists and surgeons. Thus, in the disease multiple adenomata of the intestine—a lesion which appears persistently in certain families—statistical evidence has shown that cancer supervenes with remarkable regularity, arising, it is believed, in one or more of the multitude of papillomata which stud the mucous membrane. Again, the shaggy villous type of papilloma, though innocent at



FIG. 455.—Portion of colon removed by operation from a case of multiple adenomata. Note the little round tumours scattered over the mucous membrane. (M.A. 5.) ( $\times \frac{1}{2}$ .)

first, is notoriously prone to assume a malignant character. The pedunculated polyp need not be regarded with the same anxiety, but even with this attention has often been called to the fact that a cap of malignant cells may later cover these projecting growths. The relationship which exists between simple and malignant tumours of the large intestine is the main theme of this article, which reports investigations undertaken to define this relationship more exactly. The subject may be presented most conveniently by considering the problem from the following points of view: (1) *The development and structure of the simple adenoma*; (2) *The association of simple with malignant tumours*; (3) *The intimate structure of early adenocarcinomata of the rectum and colon.*

## 1. THE DEVELOPMENT AND STRUCTURE OF SIMPLE ADENOMATA.

*Fig. 455* shows a portion of the colon in a case of multiple adenomata. The whole large intestine from caecum to sigmoid was studded with innumerable smooth rounded tumours, most of them about 1 mm. in diameter, a few pedunculated but the majority sessile. No cancer was present. The patient,



FIG. 456.—Portion of colon removed by operation from a case of multiple polypi. ( $\times 1$ .)

a young woman of 31, belonged to a family in which multiple adenomatosis and cancer of the bowel were exceptionally common.\* *Fig. 456* is from a case of multiple polypi of the colon. These two pictures serve to show the naked-eye character of the material whose intimate structure has been studied in serial sections cut through the tumours and surrounding intestine. My views are based on an examination of several portions of the intestine of three cases of multiple adenomata and an examination of more than sixty specimens of adenomata, polypi, and villous tumours sent for laboratory examination at St. Mark's Hospital during the last four years.



FIG. 457.—Section through a very early tumour from a case of multiple adenomata. Note small area of epithelial hyperplasia 'A' distinguished by cells with deeply-staining nuclei; also forward bending of muscularis mucosæ. (*M.A.* 5.) ( $\times 8$ .)

Adenomata arise from small areas of increased epithelial growth which can be distinguished under the microscope by the fact that the cells secrete

\* Mr. Lockhart-Mummery, who operated on this patient, has recently published her family history in a paper entitled "Cancer and Heredity", *Lancet*, 1925, i, 427.

less mucus than their neighbours and their nuclei stain more deeply with hæmatoxylin. This condition is well shown in *Fig. 457*. The more active growth of the epithelial coat results in a projection into the lumen of the bowel, accompanied by a bowing of the attached coat of muscularis mucosæ. *Fig. 458* shows two such early tumours.

The various stages in the growth of adenomata, whose mature form will be illustrated in subsequent photographs, is represented in *Figs. 459-463*.

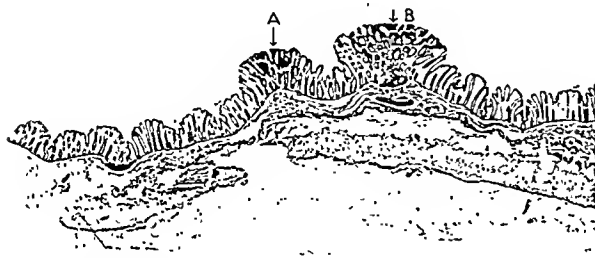


FIG. 458.—Another section through two early tumours from same case as *Fig. 457*. A collection of lymphoid cells obscures the bending of the muscularis mucosæ below the larger tumour. A, B, Early adenomata. ( $\times 8$ .)

Here the secreting cells of the large intestine are depicted as a folded coat closely adherent to the muscularis mucosæ and the scaffolding of reticular tissue, muscle, and vessels which the muscularis mucosæ supplies to the spaces between the crypts. If the epithelial layer between the points marked 1 and 7 in *Fig. 459* grows more rapidly than neighbouring districts, the first result must be an increase in depth of the crypts, accompanied by an elongation of the scaffolding which rises at right angles to the muscularis foundation, a state of affairs indicated in *Fig. 460*. This achieves a slight increase in secreting area. Should the impulse to more active growth or function continue to act in the same region, then the first stage of tumour formation proper must occur, the essential features of which are, as is shown in *Fig. 461*, a forward bending of the muscularis mucosæ in order to provide a more extensive base to support the growth, and a further lengthening and branching of the scaffolding structure. The assumption of this form enables the same foundation of bowel wall to accommodate a greatly increased surface of secreting cells, but this advantage is purchased at the cost of serious injury to the hitherto unaffected cells situated at the boundary of the young tumour. *Fig. 462* shows that because of the compression to which these marginal cells are exposed the crypts become stunted, and the constant friction leads to 'collar catarrh': of this important consequence of tumour growth more will be said later. *Fig. 463* depicts the further growth of an adenoma whereby it becomes pedunculated. These diagrams speak for themselves, and I need do no more than call attention to the stunted growth at the collar of the tumour, to the continued branching of old and appearance of new scaffolding, to the almost inevitable formation of cystic spaces,

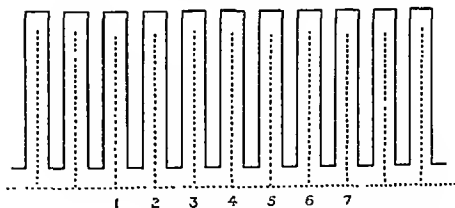


FIG. 459.—Diagrammatic representation of normal mucous membrane, the secreting cells figured by the continuous lines and the muscularis mucosæ and reticular tissue by the dotted lines.

and finally to the fact that secondary changes may begin to appear in the mucous membrane adjoining the primary tumour.

This description of the mechanics of tumour formation is built upon the supposition that the only operative factor is a more lively multiplication of epithelial cells within a defined area. It is opposed to the view that adenomata owe their origin to some submucous structure such as a lymphoid follicle bulging the secreting cells into the lumen of the gut, of which I found no evidence. Chronic inflammation is, of course, a frequent precursor of tumour growth, but if any such antecedent influence had acted in the tumours I have studied, it left no permanent imprint on the tissues.

In view of what will be said in the succeeding section on the association of simple with malignant tumours, it will prepare the ground to record the frequency of adenomata in the intestines of patients dying from diseases other than cancer of the large bowel. Thanks to facilities granted by University College Hospital, the Middlesex Hospital, and the Cancer Hospital, I have been able to examine 127 intestines which I have searched for simple tumours from cæcum to sigmoid. One or more tumours were found at some point in the bowel in 12 of these, which indicates a distribution of 9.4 per cent. In one patient 6 tumours were present, in one 4, in one 2, and the other nine contained only an isolated polyp. Of the 12 patients, 4 were women and 8 men. The average age of death amongst the patients with tumours was 46 years, and amongst the patients free from tumours 38 years, which gives support to the view that intestinal tumours are more frequent in later years.

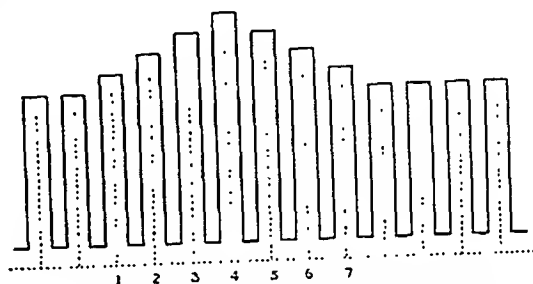


FIG. 460.—First stage in epithelial hyperplasia. Deepening of crypts and lengthening of villi.

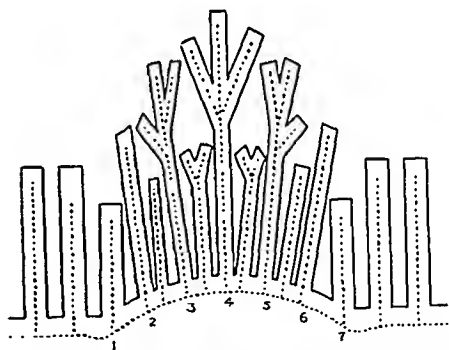


FIG. 461.—First stage in tumour formation. Bending of muscularis mucosae, branching of original villi, and birth of new villi: stunted growth at margin.

## 2. ADENOMATA ASSOCIATED WITH CANCER.

In the last 33 consecutive cases of cancer of the rectum and sigmoid at St. Mark's Hospital I have made a special search for adenomata in the portion of the bowel removed at operation, and found these tumours in 25 cases. These little tumours are easily seen before the piece of bowel is hardened in fixative solution, but the shrinkage and distortion caused by formalin make them much less easy to find. They are demonstrated most clearly if in the

fresh specimen the mucous membrane and submucosa are dissected off the muscle wall immediately; then they stand out prominently as little hillocks on the plane surface surrounding the cancer. Some are similar to the little

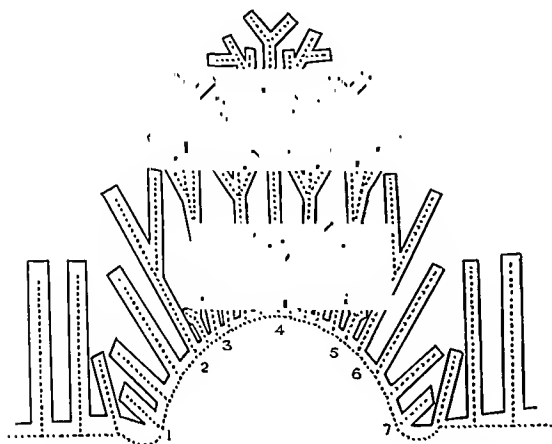


FIG. 462.—Stago intermediate between non-pedunculated and pedunculated tumour. Note increased bending of muscularis mucosæ and further branching of villi, leading to a great increase in secreting area. 'Collar catarrh' would be established at this stage.

This series of cases is not strictly speaking comparable with the investigation reported above, whereby tumours were found in 9.4 per cent of patients dying from disease other than cancer of the bowel, because in studying the normal distribution of adenomata the whole intestine from cæcum to sigmoid was examined after the tissues had been hardened in fixative, whereas in the cancer series only a few inches of bowel around the cancer were examined in the fresh condition. Accepting these reservations, both sets of figures are of considerable interest and lead to the conclusion that well-developed adenomata

tumours, about one millimetre in diameter, familiar in multiple adenomata; others grow to large pedunculated polypi. Within a radius of three inches from the margin of the cancer a few of these tumours can nearly always be found if the specimen is examined in the way described. The before-mentioned frequency of 75 per cent (25 cases out of 33) is probably too low an estimate, because, by the surgical operation—whether perineal excision or even abdominoperineal—only a few inches of intestine are removed above and below the growth, and I have not had an opportunity of examining the rest of the bowel in these cases.

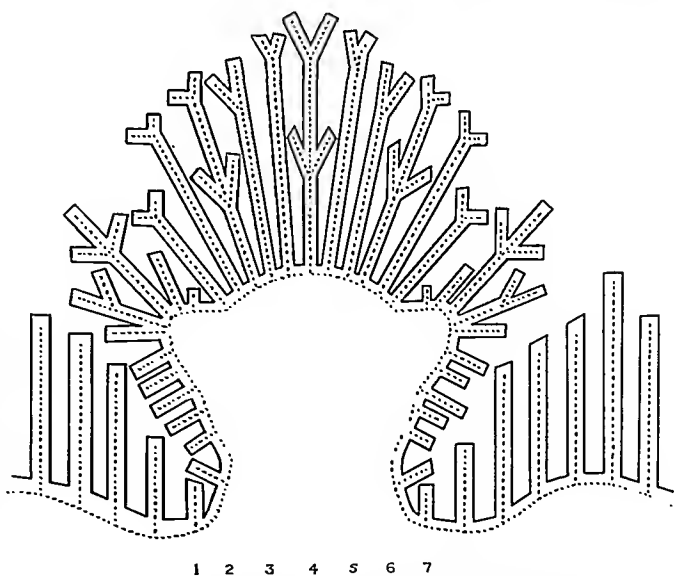


FIG. 463.—Second stage in tumour formation, showing development of stalk. Note stunted growth at margin, leading to 'collar catarrh'. The bending of the muscularis mucosæ on either side points to the development of secondary tumours which will surround the primary.

lead to the conclusion that well-developed adenomata



are found in the portion of bowel between caecum and sigmoid in only about



FIG. 464.—To show the naked-eye appearance of the simple tumours associated with a cancer of the rectum.

10 per cent of the population, whereas little tumours are almost invariably present in the mucous membrane surrounding a cancer of the rectum or sigmoid.

In order to gain further light on the relationship which exists between simple and malignant tumours, serial sections were cut through the cancers, through the neighbouring mucous membrane, and through the associated adenomata. The conclusions reached from a study of sections through the cancer may be left to the third part of this paper, which treats of the adenomatous structure of cancer: here only the surrounding mucous membrane and associated adenomata will be described.

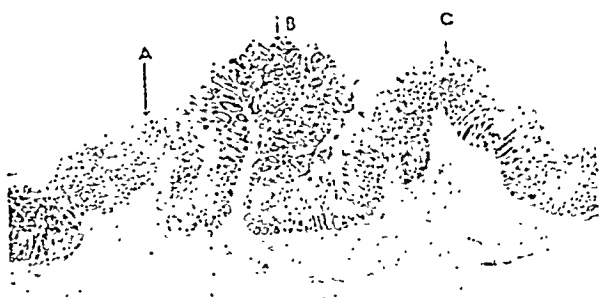


FIG. 465.—Section through a small pedunculated adenoma, one of twelve tumours arising from the mucous membrane in the neighbourhood of a cancer of the rectum. Secondary tumours (A, C) are developing on either side of the primary pedunculated tumour (B). Note 'collar catarrh' in groove between primary and secondary adenomata. Same case as Figs. 466-470. (M.A. 3.) ( $\times 8$ .)

Fig. 464 is reproduced to show the naked-eye characters of the adenomata or papillomata surrounding a cancer of the rectum. In a case similar to

this twelve tumours varying from 1 to 3 mm. in diameter were found in the three inches of mucous membrane surrounding the cancer, and these were all removed for examination together with several pieces of mucous membrane of natural appearance. The low-magnification photographs (*Figs. 465-471*) illustrate the following features to which I wish to draw attention:—



FIG. 466.—Section from another pedunculated adenoma from same case as *Fig. 465*. Note: A, Great irregularity of neighbouring mucous membrane giving birth to secondary tumours; and B, Primary adenoma with 'collar catarrh'. (*M.A. 3.*) ( $\times 8$ ).

1. These tumours are essentially similar in structure to those described in the previous section: they consist of a central stroma of connective tissue with dilated blood-vessels and are covered by a thick layer of columnar epithelial cells.

2. Round the neck of the tumour the epithelial proliferation is hampered, and in the sulcus which separates the tumour from the surrounding mucous membrane a chronic catarrh exists, a condition which I have described as 'collar catarrh' (*Figs. 465-468, and 470*).

3. Owing probably to this irritation other tumours arise on either side, so that a circle of secondary tumours comes to surround the primary (*Figs. 465-467, and 470*).

4. When the increased epithelial growth affects an area greater than can be compensated for by a projecting growth, a folding of the mucous membrane follows, and some secreting cells are pushed into the region of the submucosa (*Figs. 467-470*).

5. Round such an infolded column of cells the normal lymphoid tissue is greatly increased and collected in conspicuous follicles, often forming a cap round the displaced secreting cells (*Figs. 467-470*).

6. Those parts of the mucous membrane in the neighbourhood of a



FIG. 467.—Section through another pedunculated adenoma from same case as *Fig. 466*. Note increase of lymphoid tissue at points where mucous membrane is folded inwards. A, Lymphoid follicle; B, Infolding of mucous membrane; C, Primary adenoma. (*M.A. 3.*) ( $\times 8$ ).

6. Those parts of the mucous membrane in the neighbourhood of a

## SIMPLE TUMOURS OF LARGE INTESTINE 727

cancer of the rectum which bear no tumours visible to the naked eye show under the microscope a curious 'switchback' appearance, as though the epithelial growth were alternately increased and diminished (*Fig. 471*). This 'switchback' appearance is very common in the mucous membrane surrounding a cancer.

Two further characteristics of these simple tumours which bear upon our problem are revealed by higher magnifications. *Fig. 472* illustrates the fact that some of the cells of an adenoma secrete mucus and others do not. *Fig. 473*, which was taken

from an actively growing adenoma in which many mitotic figures were found



*FIG. 468.*—Another section through the same tumour as *Fig. 167*. A, Primary adenoma; B, Cap of lymphoid tissue covering inward folds of mucous membrane. ( $\times 8$ .)



*FIG. 469.*—A higher magnification of the same slide from which *Fig. 468* was taken, to show cap of lymphoid tissue (A) covering inward folds of mucous membrane. ( $\times 24$ .)

within the epithelial cells, illustrates the fact that, when branching of the stroma cannot keep pace with the continued epithelial hyperplasia, then the columnar cells become stratified several layers deep or may even sprout out into the lumen, forming miniature polypi within the duets. The particular tumour shown in *Fig. 473* was remarkable not only for the mitotic figures within the epithelial cells, but also because of a barrier of lymphoid tissue which separated the deeper cells from the muscularis mucosæ, in which also mitotic figures were plentiful (*Fig. 474*). Mitotic figures are only very rarely found in the epithelial cells of adenomata surrounding a cancer.

From the point of view of the relationship of benign to malignant tumours of the bowel, a question of paramount importance is the order in which these growths appear: did the simple tumours precede the cancer or have they developed subsequent to the malignant growth? Some indirect

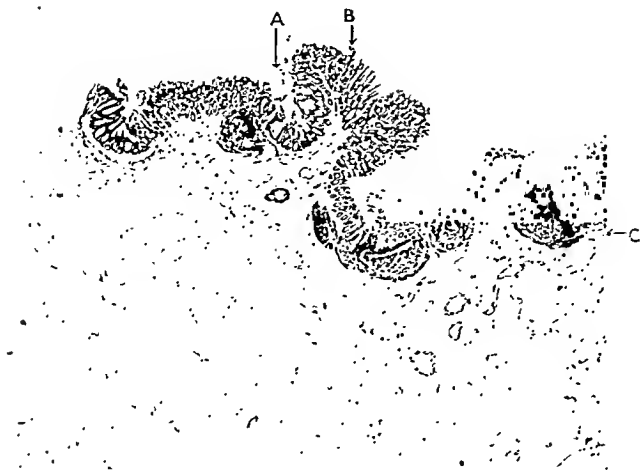


FIG. 470.—Section through another tumour from the same case. Note cystic dilatation of the glands, 'collar entorrh', and great increase of lymphoid tissue. A, 'Collar entorrh'; B, Adenoma; C, Lymphoid follicle. (M.A. 3.) ( $\times 8$ .)

FIG. 471.—Section through a portion of the mucous membrane of the rectum in a case of cancer of the rectum accompanied by six small adenomata within three inches of the cancer. Note 'switchback appearance' of the mucous membrane, which here bears no tumours visible to the naked eye. (M.A. 11.) ( $\times 4$ .)

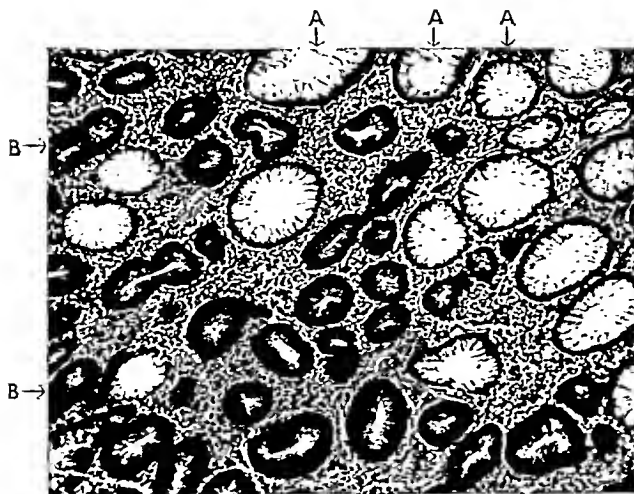


FIG. 472.—Transverse section through one of the six simple tumours in same case as Fig. 471. Note that some cells are secreting mucus (A) and others are not (B). (M.A. 11.) ( $\times 90$ .)

evidence on this question of seniority can be obtained from a consideration of the general structure of adenomata and the morphology of the constituent cells. A firm pedunculated adenoma, the cells of which are vigorously secreting mucus, has had time to consolidate its position and cannot be regarded as a very recent growth. Cells exhibiting many mitotic figures, on the other hand, indicate a district where rapid growth of the tumour continued to the moment of extirpation. Intermediate in age between the cells undergoing mitosis and the goblet cells loaded with mucus we can recognize the type of cell with large deeply-staining nucleus not secreting any mucus at all.

Information gained from the morphology and behaviour of the cell cannot, of course, fix the age of the tumour exactly, but it permits of a rough classification into tumours still

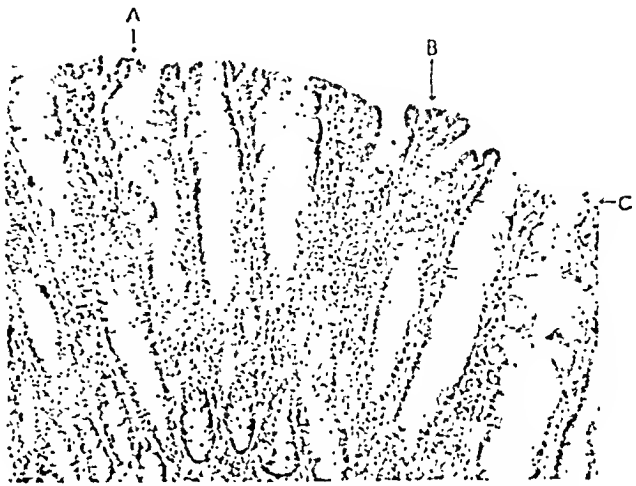


FIG. 173. Section through a small area of epithelium, growing rapidly and just commencing to form a small tumour, situated close to a cancer of the rectum. The epithelial cells were not secreting mucus and showed numerous mitotic figures. Note pleating (B) and stratification (A) of the epithelium and formation of miniature interglandular polypi (C). A barrier of lymphoid tissue (see Fig. 171) separated the deeper cells from the submucosa. (M.A. 14.) ( $\times 75$ .)

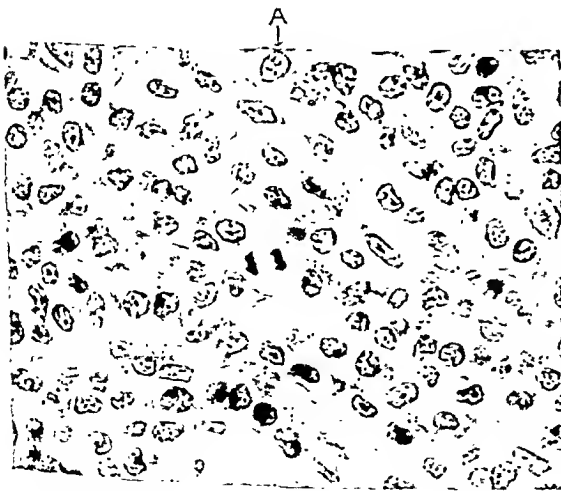


FIG. 174.—Section through barrier of lymphoid tissue beneath rapidly growing epithelium shown in Fig. 173. Note mitotic figures in lymphoid cells in centre of photograph in the direction indicated by A. (M.A. 14.) ( $\times 665$ .)

actively growing, indicated by mitotic figures; tumours in which growth has recently occurred but is not now taking place, indicated by cells with large deeply-staining nuclei; and mature adenomata whose cells resemble the neighbouring mucous membrane both in form and function. When these tests are applied to the adenomata which accompany cancer of the rectum and sigmoid we find that most of the adenomata consist of a mixture of cells some of which are secreting mucus and others not (Fig. 172).

Glands lined with cells actively secreting mucus adjoin other tubes lined with

closely packed mucus-free cells, distinguished by large deeply-staining nuclei.

In such tumours mitotic figures cannot be found, and the cells appear to have passed beyond the initial stage of lively growth and are now commencing to accommodate themselves to their appropriate function. Well-established pedunculated polypi, on the other hand, are usually covered with a layer of cells all actively secreting mucus. Tumours that are composed entirely of non-mucus-secreting cells are very rare, and as for mitotic figures, these are practically never found.

This evidence may be summarized by saying that most of the simple tumours surrounding a cancer have passed beyond the initial stage of lively growth, and that in the non-pedunculated tumours some of the epithelial cells are beginning to secrete and others are already at work secreting mucus, and that well established pedunculated adenomata are also common round a cancer.

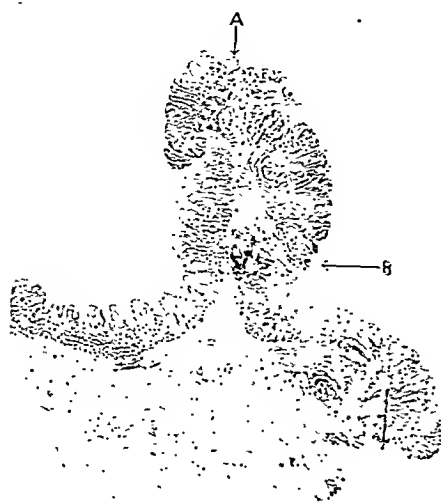


FIG. 475.—A polypoid adenoma situated close to a large ulcerating cancer of the rectum, but separated from it by an inch of normal mucous membrane. Other smaller adenomata were also present. The cells at the tip of this tumour had undergone cancerous degeneration (A) (see Fig. 476); B, Lymphoid follicle in stalk of tumour (dark mass). (M.A. 9.) ( $\times 6$ .)

### 3. THE INTIMATE STRUCTURE OF EARLY ADENOCARCINOMATA.

The majority of cancers of the large bowel removed by operation have the character of large projecting masses, or shallow ulcers with raised edges. Because of the general confusion caused by the irregular growth, and also because of infection with bacteria of the faeces leading to inflammatory reaction, little can be learnt from these mature tumours about the initial architecture of the growth.

Very early cancers, however, provide instructive material for study, and two of the cases in this series came within that category.

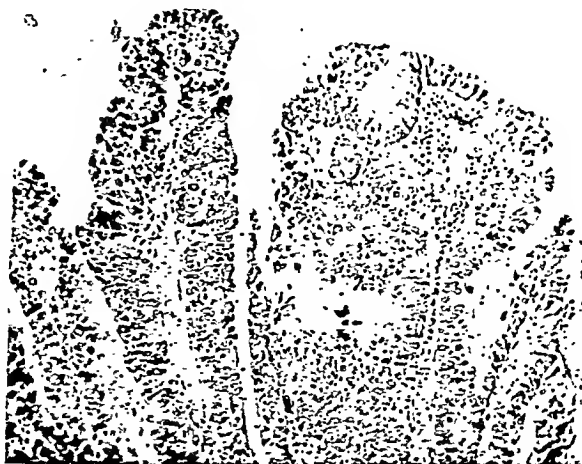


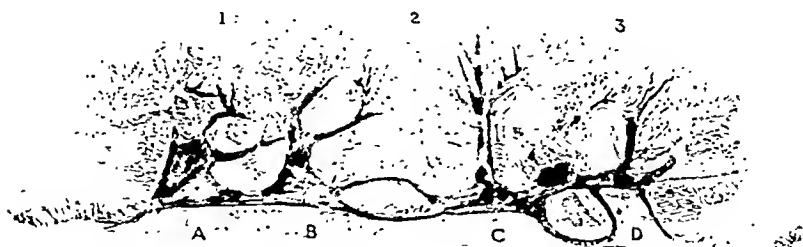
FIG. 476.—A higher magnification of the tip of the polypoid adenoma illustrated in Fig. 475, to show onset of cancer at tip. ( $\times 135$ .)

*Fig. 475* represents a large pedunculated adenoma situated above an ulcerating cancer of the rectum and separated from it by an inch of normal mucous membrane. Other smaller adenomata were also found round about, but this tumour was of special interest because it had already undergone malignant degeneration at the tip, as shown by *Fig. 476*. Though cancerous



*FIG. 477.*—A complete section through a very early cancer of the rectum, which measured only 1 cm. in its broadest diameter and projected like a little button about 3 mm. above the surface of the bowel. Four small non-pedunculated adenomata were found within a radius of two inches of the growth. (*M.A.* 6.) ( $\times 8$ )

at the tip, the mucous membrane covering the neck of the polyp showed no evidence of cancer. Obviously this was an instance of cancer developing in an established benign adenoma. Similar cases to this have been described and illustrated by Dr. Verse.<sup>1</sup>



*FIG. 478.*—The same section as *Fig. 477*. Whilst the slide was under the microscope the supporting tissue in the photograph was painted a deeper colour, thereby emphasizing the fact that essentially the cancer has the structure of four adjacent pedunculated tumours, A, B, C, and D, between which the cancer cells have penetrated to their greatest depth. On the surface at the points marked 1, 2, and 3, the slight depressions may represent the original clefts between the adjacent tumours. ( $\times 8$ )

*Fig. 477* is an enlargement of a complete section through the middle of a very early cancer of the rectum. The tumour measured only 1 cm. in its broadest diameter, and projected like a little button about 3 mm. above the surface of the bowel. Four small non-pedunculated adenomata were found within a radius of two inches of the growth. Serial sections were cut

through the entire cancer and underlying bowel wall, and the general architecture of the tumour was reconstructed from these. In its central part the tumour consisted of four stout trunks of stroma rising from a thickened base. Whilst the slide was under the microscope this supporting tissue was painted a deeper colour, as in *Fig. 478*, thereby emphasizing the fact that essentially the cancer has the structure of four adjacent independent tumours, A, B, C and D, between which the cancer cells have penetrated to their greatest depth. On the surface, at the points marked 1, 2, and 3, the slight depressions may represent the original clefts between the adjacent tumours. I have already referred to the fact that a simple adenoma of the rectum may later become surrounded by a circle of secondary tumours (*see Figs. 465, 466*), and if the district of increased epithelial growth affects an area greater than can be compensated for by forward bulging, then the mucous membrane must become folded on itself (*see Figs. 467, 468*). With these considerations in mind I formed the conclusion that the cancer represented in *Figs. 477 and 478* arose from one of the many adenomata scattered over the mucous membrane. The primary tumour became surrounded by secondary adenomata. Later, malignant changes developed in the cells between the adjacent tumours, which, because of their position, were restricted in their growth and suffered from 'collar catarrh'.

### GENERAL CONCLUSIONS.

This study of simple and malignant tumours of the large bowel brings the pathology of cancer of this region into line with recent views as to the early stages of cancer in other regions of the body, such as the views of Sir Lenthal Cheate on the early stages of cancer of the breast.<sup>2</sup>

Strong support for the view that cancers of the bowel develop in preceeding simple adenomata is offered by recent experimental work on the production of tar cancer in mice. Dr. J. A. Murray's<sup>3</sup> experimental researches have shown that, after painting the skin of laboratory animals with tar, tumour growth begins in isolated, usually minute areas, and not diffusely through the whole zone of application of tar. Dr. H. T. Deelman<sup>4</sup> observed that after about three months' regular application of tar to the shaved skin of mice a crop of little tumours arose, almost without exception, on the painted area. These varied in number from three to twenty and were generally between one and two millimetres in diameter; some remained small, and others developed to pedunculated papillomata. Judging from the series of photographs of these little tumours with which Dr. Deelman's article is illustrated, it would seem that these experimental tumours are identical in appearance with the simple adenomata which accompany a cancer of the bowel. Cancerous degeneration overtook one or other of these experimental simple tumours usually between six and ten weeks after the scattered irregularities of the painted surface were first noticed; after the cancer had developed the remaining simple tumours progressed no further.

With the experience gained from tar cancer as a guide we can figure the order of events in the development of a cancer of the rectum as follows. The first effect of the carcinogenic agent is that the epithelial cells of the mucous



membrane covering an extensive area of the bowel are stimulated to more vigorous growth in *many separated spots*, a state of affairs which may be recognized microscopically even before irregularity of contour can be distinguished by the naked eye. Later, *a crop of adenomata arise from this sensitive field* and some of these become surrounded by secondary tumours. The mucous membrane in the clefts between primary and secondary tumours is hampered in its growth and becomes folded inwards (*see Figs. 465-469*). Possibly these dislocated cells are the first to undergo malignant changes. But whether this be so or not, there is no doubt that the cells in the space between primary and secondary tumours are mechanically irritated, for in the condition I have called 'collar catarrh' there is abundant evidence of chronic irritation.

*Why* cancer develops is an extremely interesting but rather unprofitable question for speculation in the present state of our ignorance. But to the questions *when* and *where* I hope these studies have made some contribution, for they have led me to the conclusion that cancer makes its appearance when a crop of simple tumours has arisen in the irritated field, and in close association with one of these.

Part of the expense of this research was met by a grant from the British Empire Cancer Campaign. I wish to thank the following pathologists for their kindness in providing me with material for study: Dr. Barnard, of University College Hospital; Dr. Baker, of the Middlesex Hospital; and Dr. Fry, of the Cancer Hospital. I am also under a special obligation to Mr. Lockhart-Mummery, Senior Surgeon to St. Mark's Hospital, for his interest in my work, and for the opportunity of consulting his opinion on several questions.

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- <sup>2</sup> CHEATLE, SIR LENTHAL, *Brit. Jour. Surg.*, viii, 149, 285; ix, 235.
- <sup>3</sup> MURRAY, J. A., *Eighth Scientific Report of Imperial Cancer Research Fund*.
- <sup>4</sup> DEELMAN, H. T., "Ueber experimentelle maligne Geschwülste durch Teereinwirkung bei Mäusen," *Zeits. f. Krebsforsch.* 1921-22, xviii, 261.

## REPORT OF A CASE OF SOLITARY FIBROCYSTIC DISEASE OF THE HUMERUS EXHIBITING SPONTANEOUS RESOLUTION: WITH A REVIEW OF THE LITERATURE AND A CONSIDERATION OF THE ETIOLOGY AND TREATMENT.

A. WILFRID ADAMS, BRISTOL.

RARE as it is, this malady has many claims on our interest. Commonly it declares itself in dramatic and forcible fashion by spontaneous fracture, suggestive of malignant disease of bone, but happily having its own unique radiographic picture. It is very amenable to operative treatment, and, indeed, may cure itself unaided if given the chance. It was this surprising finale in the case about to be recorded that led to a search of the literature and to the suggestion of factors concerned with the etiology of fibrocystic disease of bone.



FIG. 479.—K. R. Fibrocystic disease of humerus (January, 1924).

### CASE REPORT.

In January, 1924, K. R., age 13 years, had pain and weakness in his right arm following a slight injury at play a few days previously. Crepitus was obtained near the upper end of the right humerus and some thickening of the shaft detected, but no bruise or deformity was present. The skiagram (*Fig. 479*) affords a good example of fibrocystic disease. A pathological fracture is to be seen in it as a transverse crack across the upper segment of the humerus, which is markedly expanded. The cortex of the bone is only a thin shell, while the enlarged medulla is decalcified and cystic. Although so near the epiphyseal line, the disease has nowhere strayed across it, but is confined to the metaphysis. The usual treatment for fracture of the humerus resulted in union, and, after two months, the boy was fitted with a light leather support allowing full shoulder movements. However, a trifling injury fractured the bone again higher up. It united, only to be refractured for the third and last time within four months. The fragments fused satisfactorily, but in the presence of such fragility eradication of the disease was considered necessary. His admission into hospital was unavoidably delayed. This proved beneficial, for a radiogram in October, 1924, showed shrinkage of the medulla and re-formation of the normal cortex. In view of this

propitious change in the train of events, operation became unnecessary. *Fig. 480* (January, 1925) shows the next step towards spontaneous resolution, and in *Fig. 481* (December, 1925) the bone is once more perfectly strong. No sort of medicinal treatment was used, but only splintage and massage in the early stages. At the present time, January, 1926, he has discarded the light leather support and has full power and unrestricted use of his arm.

Examination of the rest of the skeleton has revealed no other focus. The Wassermann reaction is negative. His own and his family history are healthy.

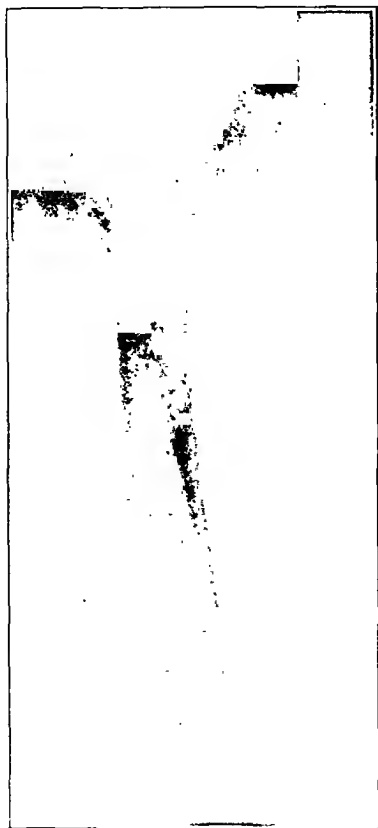


FIG. 480.—K. R. Fibrocystic disease of humerus (January, 1925).



FIG. 481.—K. R. Fibrocystic disease of humerus (December, 1925).

## GENERAL OBSERVATIONS.

**Pathology.**—Before discussing its etiology, a clear view of the structural changes found in this disease is desirable. It is notable that, as in many osseous disorders, there is a tendency for fibrocystic disease to affect the skeleton in three degrees. Thus it may involve one or many bones, or give rise to a diffuse affection of the whole osseous system. Like the common cancellous exostosis, it is usually a solitary lesion at the end of one of the long bones, and so contrasts sharply with a disease such as osteitis deformans, where a widespread affection of the whole skeleton is the rule. In this contribution interest is focused on the solitary simple fibrocystic lesion.

As no operation was performed in the case here recorded, the histological report so clearly given by Silver<sup>1</sup> on the morbid material derived from his own allied case is quoted as concise and typical:—

“The bone trabeculae . . . are separated by a fibrogranulomatous tissue

consisting of long and short spindle-shaped cells. . . . The bone trabeculae are lined with one or more layers of osteoblasts and in some places are groups of osteoblasts". The fibrocystic area occupies the medulla of the bone. It is lined by membrane in some cases, and may contain, in addition to the common fibrous and fluid elements, cartilaginous, bony, myelomatous, or even sarcomatous tissues. As the cyst expands and causes absorption of the cortical bone, a pathological fracture is a common complication, and incidentally the reason for which the patient usually first seeks the doctor. The clinical records of these cases often contain recurrent fractures and stretch over a long period of years, until finally an operation such as thorough curettage exterminates the disease.

During the history of fibrocystic swellings of bones much has been written on its relationship to other osseous dystrophies, and this received attention recently from Dawson and Struthers,<sup>2</sup> who state in their monograph: "There is good reason for believing that the pathogenesis of rickets, osteomalacia, osteitis fibrosa and osteitis deformans is allied, and it is recognized that the pathological conceptions of these diseases pass over into one another". Indeed, they consider that the histology of osteitis fibrosa and osteitis deformans is 'identical', but note that the latter occurs later in life and the bones do not break but bend.

**Etiology.**—The disease seems to have been given public definition first in 1891, when von Recklinghausen gave a description of the diffuse form which has become classical. He pronounced it an inflammatory lesion. Spontaneous resolution such as occurred in the present case would be compatible with this view, whilst on the other hand it certainly seems to negative Virchow's older teaching which attributed bone cysts to cystic degeneration in chondromata. If the fibrocystic condition is an inflammatory reaction, it does not appear to be provoked by a stimulus of an infective nature, for no mention is made of any bacterium or parasite being found in any of the cases in all the records at my disposal. Staphylococci were present in one of the cases of tibial cyst reported by Elmslie,<sup>3</sup> but were regarded as due to skin contamination. Certainly from the skiagraphic standpoint one does not associate with infective osteitis the very smooth, thin, and sharply-defined bony shell which typifies bone cyst. Lubarsh and Rabke are quoted by Young and Cooperman,<sup>4</sup> writing on the diffuse disease, as protagonists of the infective theory, but Morton<sup>5</sup> in discussing etiology states that the condition is certainly not due to any of the ordinary bacteria. Lawford Knaggs<sup>6</sup> argues in favour of osteitis fibrosa, osteitis deformans, and osteomalacia being reactions of different degrees to the action of toxins on the skeleton.

Evidence in favour of the endocrine origin of the disease is weighty and growing. Young and Cooperman<sup>4</sup> refer to Boit's view that the changes in the bones result from toxic, metabolic, or infectious processes, and also derangements of internal secretions. Morton<sup>5</sup> quotes cases of an associated parathyroid tumour having been found by Meyer in one case and similar findings by Schmorl in closely related forms of disease, adding that Meyer thought it accidental. The most tangible support comes from the case of multiple fibrocystic disease reported by Dawson and Struthers,<sup>2</sup> involving

gross decalcification of many bones and in which a parathyroid tumour the size of a cherry was found in addition to the natural representatives of that gland. Their case was accompanied by a pathological deposit of calcium salts in the muscles, and they conclude their discussion on the etiology of osteitis fibrosa with the words. "The association of parathyroid abnormality with two extreme instances of calcium metabolic disorder is very suggestive of an endocrine origin". The foregoing writers have been concerned, it appears, mainly with the multiple and diffuse varieties of the disease.

In the past trauma has figured sufficiently as the causative factor in this pathological process to merit reference. Von Miekulicz in 1904 opposed the inflammatory theory of von Recklinghausen by attributing these cysts of bone to a 'simple' disturbance of growth. Bloodgood<sup>7</sup> in 1910 expressed his opinion, "I therefore have been unable to find any definite etiological factor, and I cannot agree with those who look upon the lesion as the result of trauma, but it cannot be denied that there is some justification in considering the possibility of traumatism as a factor". Elmslie,<sup>3</sup> in reviewing the present situation (1914) on the nature of bone cysts, writes: ". . . and they have been recorded by Frankenhelm in callus at the seat of a fracture". David Silver<sup>1</sup> in his remarks on etiology includes traumatic as the second of the theories given. He quotes the opinion of Beneke that bone cysts are analogous to apoplectic cysts of the brain, and notes that if they are simply localized osteitis fibrosa, then that in turn may itself own a traumatic origin. A memorable warning is sounded by Elmslie in this connection as follows: "Next to fracture, pain and swelling are the most frequent symptoms. These may arise spontaneously; more often they come on after an injury. Some observers have on this account ascribed a traumatic origin to the cysts. It appears more probable that the injury has caused an incomplete fracture through a pre-existing cyst, and that pain and swelling have resulted from further enlargement of the cavity". On these grounds one assumes the existence of a fibrocystic focus prior to the forcible twisting of the arm in Heath's<sup>8</sup> case. In 1922, Young and Cooperman<sup>4</sup> conclude that "traumatism does not cause the general form of this disease, although there is distinct evidence to show that local osteitis fibrosa and benign bone cysts are caused by trauma". Although the foregoing evidence is too slender a support on which to argue in favour of a violent origin for solitary fibrocystic disease, the writer feels that in the form of the repeated minor stresses of active life, the mechanical factor of force does appear to be a contributory exciting factor in the pathogeny of fibrocystic disease.

On investigating the site and age incidence of cysts in the solitary variety of the disease, one finds the bulk of the cases concentrated in a few regions of the skeleton, and that almost all of them start in youth. Bloodgood collected 57 cases of solitary true bone cysts, and states, without giving detailed statistics, that "benign bone cysts are found prominently in the humerus, femur, and tibia (in that order of frequency). More often they extend to the epiphyseal line, but there are cases in which the disease involves the centre of the shaft only". As to age, he asserts that "the disease with the rarest exceptions starts before twenty—that is, before complete ossification of the epiphysis". Silver gives figures, but does not specify the end of

the bone affected in his cases. He gives the order of frequency as femur, humerus, tibia. If we confine ourselves to the figures of Elmslie's cases in which alone the particular extremities are mentioned, we find these startling proportions :—

Humerus, upper end	21 = 35	per cent
„ lower end	3 = 5	„
Femur, upper end	13 = 21	„
„ lower end	10 = 16	„
Tibia .. ..	7	
Other bones ..	5	
<hr/>		
Total	59	

Four of his cases affecting the tibia in which the condition appears not quite typical have been omitted, leaving 59. If to these are added the case recorded by Heath and the present one, it merely emphasizes the striking preference of the disease for the upper end of the humerus and ends of the femur, where epiphyseal plates appear early, work late, and with great rapidity. Two factors combine to distinguish these sites—namely, marked cellular activity and great stress. First, it is at them that the most rapid deposition of bone takes place during growing years. Secondly, on them, too, unusual stresses fall owing to the transmission of rotary movements near ball-and-socket joints and the action of strong muscles of the great body-girdles pulling on the humerus and femur in all directions. Additionally, in the leg is the vertical pressure of body weight. How real is the rotary stress to which the bone-end is subjected is shown by the fact that nature guards against the tendency for the head of the humerus to twist round on the shaft by developing conical bosses at the upper end of the diaphysis on which the head of the bone is securely seated (*Fig. 482*). The vulnerable nature of the delicate formative tissues of this juxta-epiphyseal region is borne out by the frequency with which disease invades it in the early years of life—for example, in rickets, congenital syphilis, acute osteomyelitis, tuberculosis, and the cancellous exostoses.

The occasional mid-shaft position of fibrocystic disease calls for explanation, as it would seem at first sight to vitiate the argument that attributes its origin to the metaphysis. Its presence in the middle of the shaft is due to its displacement thither when the influence that provoked its formation has ceased to act, and the bone-cell factory at the epiphyseal line has resumed the supply of healthy bone. Thus, in its mode of origin and subsequent fate it may be compared helpfully with the transverse markings visible in the finger-nails of some patients after a debilitating illness. The actual site of formation of this defective zone in the nail is up under the cuticle, but it migrates distally with the passage of time (owing to the continuous proliferation of nail proximal to it). In the series of skiagrams of the cases of E. O. and C. R. reported by Elmslie we actually see this migration from one end down the shaft of the bone taking place in the humerus. The skiagrams were taken over a period of three to four years. In fact it would seem that from the number of centimetres between the bone-end and the site of disease, the age of the patient when the metaphysis was attacked might be deduced;

but because the disease happens to occupy a diaphyseal situation when detected, it must not be taken to mean that it has always been so.

If it be granted, then, that trauma, in the sense of the constantly repeated stresses natural to the wielding of limbs, is the exciting cause and that the site of incidence is where stresses fall most on young metaphysial tissues, there still remains for explanation the rare occurrence of the disease.

From the foregoing review of the literature of this subject one is led to attribute the malign influence predisposing to fibrocystic metaplasia of bone tissue to error in the functions of the parathyroid glands, which are known to be



FIG. 482.—Two humeri prepared to show the concavity of the head epiphysis fitting the diaphyseal bosses.

closely associated with calcium metabolism. The vulnerable tissue at the metaphysis may be compared with gunpowder, and the physical stresses to which it is exposed are like sparks. Normally the two associate, but are insured against interaction and explosion by the damping effect of the parathyroid secretion. If this damper is defective, the inevitable conflagration follows and smoulders until it is cleared away by the surgeon or until an adequate supply of parathyroid secretion overcomes the flames and allows internal reconstruction to take place. This weakness on the part of the parathyroid secretion manifests itself in various grades, and may be transient or persistent. In mild

cases—for example, when a solitary fibrocystic lesion develops—it is probable that the covering influence of the parathyroid gland is barely adequate, so that at some juncture in a vulnerable area like the shoulder, knee, or hip exposed to heavy mechanical stresses, the previously latent inadequacy becomes manifest in disorganization of calcification, and a segment of bone is turned out defectively until the healthy balance of internal secretion is restored and subsequent metaphysial additions are normal. The zone of fibrocystic disease is a monument to a period of defective calcification at a certain time in an individual's growth.

These faults in the bony strata may happen more often than we suspect, in some cases giving rise to slight or even no symptoms at all, and disappearing, leaving no trace. The reports of the disease at my disposal show that the fibrocystic focus tends to persist and demands for its cure a thorough removal by curettage. This is not always so, however, and from the following facts it is fair to deduce that the underlying causal condition may act only temporarily, and then give place to an overcoming corrective influence. Silver records 6 instances of spontaneous cures, and, in the discussion following his paper, Dr. Colvin reported another, which with my own patient (K. R.) makes a total of 8 such cases. Thus among some 200 cases of monosseous fibrocystic disease about which I have information, apparently 4 per cent healed without surgical intervention. This agreeable feature allies the disease with another disorder of bone—namely, rickets. Apparently when in fibrocystic disease the parathyroid function regains its healthy level it may resume its covering influence on the affected osseous tissues. As a result the disorder in the metaphysis disappears and the cells 'harden' to withstand the physical stress they are normally called upon to bear. Fibrocystic disease seems to form one of the many guises in which defective calcium metabolism manifests itself. The phasic variation in the stability of body calcium is one of the great romances of physiology, seen in such conditions as fragilitas ossium, rickets, tetany, dental caries of adolescence and pregnancy, osteomalacia, and osteitis deformans of later life.

**Treatment.**—Finally a word is called for in connection with treatment on which the foregoing observations have a bearing. The treatment of curettage without bone-grafting advocated by Bloodgood does not appear to have been surpassed, but before deciding to operate one should not forget the historic evidence of cures that have occurred by mere external support and restrained movement continued for a sufficient length of time. The possible benefit of concurrent parathyroid feeding calls for consideration.

### SUMMARY.

1. Three factors enter into the etiology of fibrocystic disease: (a) Vulnerable formative tissues of the metaphysis, especially at the upper end of the humerus and the extremities of the femur; (b) The site of maximum physical stress on bones—that is, near the shoulder, hip, and knee where strong muscles pull and twist; (c) Instability of calcium control owing to defective parathyroid function.



2. The varying degree to which the skeleton is affected by fibroeystic disease admits of easy explanation. Thus in the mildest cases there will be a temporary disorder affecting one of the particularly susceptible regions—for example, the upper end of the humerus—and subsequent disappearance of the disease; in a moderate deficiency of parathyroid function a solitary lesion occurs and will persist; while in a severe disturbance multiple foci would manifest themselves; and finally the diffusion of the disease throughout all the bones (von Recklinghausen's disease of bones) may be regarded as a malignant form.

3. In the monosseous type of fibroeystic disease of bone spontaneous cure has only been known to occur in about 4 per cent of cases. It remains to be seen whether this percentage can be augmented by the wider adoption of palliative treatment.

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- <sup>3</sup> ELSLIE, *Brit. Jour. Surg.*, 1914, ii, 17.
- <sup>4</sup> YOUNG and COOPERMAN, *Ann. of Surg.*, lxxv, 171.
- <sup>5</sup> MORTON, *Arch. of Surg.*, 1922, iv.
- <sup>6</sup> KNAGGS, LAWFORD, *Brit. Jour. Surg.*, 1925, xiii, 230.
- <sup>7</sup> BLOODGOOD, *Ann. of Surg.*, 1910, lii, 45.
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## FAT NECROSIS OF THE BREAST, WITH AN ACCOUNT OF A CASE.

BY GEOFFREY HADFIELD, BRISTOL.

INFLAMMATORY processes in breast fat are frequently encountered in the routine examination of excised breast tissue. In the large majority of cases the gross and microscopic characters of the process are identical with those found in inflamed fat in other situations. In a few cases, however, when the injurious agent is trauma inflicted locally on a pendulous breast heavily loaded with fat, a circumscribed lesion may arise which bears a striking naked-eye resemblance to cancer, and may present several of the classical signs. Lee and Adair<sup>1</sup> first drew attention to this innocent condition in 1920, and they have now published five cases. One case has been reported by Keynes,<sup>2</sup> and Rowntree<sup>3</sup> has described one almost certainly of the same nature. Of these seven cases, four so closely resembled breast cancer that needless amputation was performed. Trauma appears to be an essential factor in the causation, a history of trauma being definite and exact, whilst its site and the location of the lesion are identical. The lesion in that stage of its evolution when it resembles cancer is hard and often adherent to the skin, sufficiently so to produce dimpling and the 'peau d'orange' appearance. In the case here reported there were two small foci of fat necrosis which, although not resembling cancer sufficiently to justify total excision, were found to bear a close resemblance to it on naked-eye examination, and microscopically showed the early stage of the lesion before the deformity and contracture of fibrosis had masked its initial phase.

### CASE REPORT.

**HISTORY.**—A woman, age 47, was admitted to hospital complaining that for the last four weeks the right breast had become painful and that a bruise-like area had appeared on its outer side. The pain was not continuous or severe, and she had felt no lump. A week before she noticed the pain a spade fell on the right breast, but the injury was slight. Her youngest child is 17; both her children were breast-fed for ten months. The breast was large, fatty, and pendulous. In the outer upper quadrant was a small, well-defined nodule which appeared to lie almost directly under the skin. It was not fixed above or below, and the skin over it was normal.

**OPERATION** (Mr. Duncan Wood).—A wedge-shaped piece of breast in the outer upper quadrant was excised. It consisted almost entirely of fat in which were two small nodules roughly circular in outline and half an inch in diameter, moderately hard and white, chalky and homogeneous in appearance. Radiating from the nodule in all directions were very fine chalky-white fasciculi. The lesions occupied the whole of a small lobule

of fat. They had produced no deformity or contracture and, except for the fine radiating fasciculi, gave the impression that a single fat lobule had become partially calcified *in situ*. Recovery was uneventful.



FIG. 483.—Microphotograph of centre of lesion. Uninjured fat globules widely separated by large foamy cells. At A a globule of fat is seen partially broken down and invaded by similar cells.

**HISTOLOGICAL EXAMINATION.**—Sections include the nodule, which lies entirely in the breast fat, two small ducts, and their related lobules. The latter are richly infiltrated by lymphocytic cells, but show little change

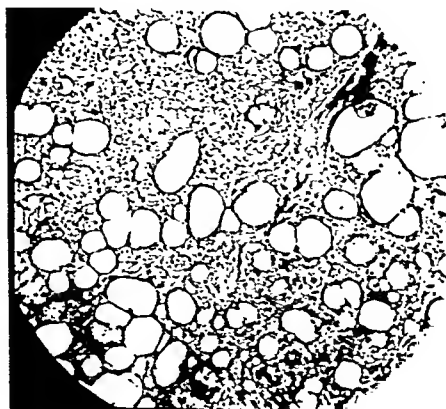


FIG. 484.—Low-power view of main mass of lesion. Uninjured globules widely separated by fat phagocytes.

beyond this. The nodule is composed of large swollen cells with foamy cytoplasm which lie between, distort, and widely separate unruptured fat globules. Its edge is fairly sharply defined, but thin fasciculi of foamy cells

spread into the surrounding fat in all directions. The lesion is almost bloodless. There is a mild fibroblastic reaction in the few connective-tissue septa which penetrate the lesion and in the neighbouring breast tissue. A few of the fat globules situated in the centre of the lesion are filled by faintly-staining material uniformly finely vacuolated or by similar material intersected by tiny cigar-shaped clefts and spaces recalling the appearance seen in such situations as the aorta where doubly-refractile cholesterol esters are deposited out of reach of living cells. In other places the outline of a fat globule can be seen, but its peripheral third is occupied by a circle of swollen cells with foamy cytoplasm and central, rather feebly staining nuclei, the whole giving the false impression of an alveolar formation. The outlines of other fat globules can only be faintly traced: these contain a central irregular finely-vacuolated mass closely invested by foamy cells, and in the centre of a few is a single multinucleated giant cell.

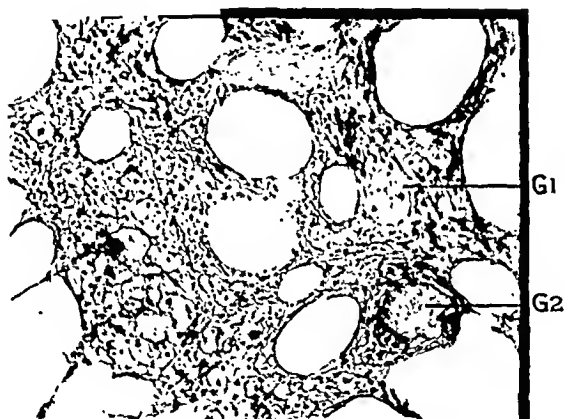


FIG. 485.—Similar changes to those seen in Fig. 483, with two unabsorbed globules, G1 and G2, still *in situ*.

The rest of the nodule is composed of unaltered fat globules and sheets of closely applied foamy cells, with a few scattered giant cells. Lymphocytic cells and normal leucocytes are absent in the centre of the lesion and scanty at its periphery, being confined almost entirely to the adjoining breast lobule. (Figs. 483-485)

### COMMENT.

The lesion hardly raised the suspicion of carcinoma on clinical examination, chiefly because of its small size, superficial situation, and the absence of signs of adhesion, the latter due to its recent formation. It was, however, difficult to exclude malignancy with certainty on naked-eye examination of the excised tissue. It is unfortunately common to find a malignant mass much larger and usually harder. The uniformly opaque appearance on section was also atypical of carcinoma, but unless fat necrosis had been

borne in mind it would have been difficult to make any diagnosis other than malignant disease in an uncommonly early stage.

The other clinical particulars of the case—for example, the history of trauma and bruising and the pendulous fatty type of breast—agree with those of Lee and Adair's cases. This case illustrates the fact that fat necrosis produces a similar clinical picture to cancer only when a sufficient interval has elapsed for replacement-fibrosis to produce contracture.

As regards the histological characters of the lesion, there seems no reason to doubt that this case represents the early stage in the genesis of the more cicatrized lesions described by other authors. Its histology is very similar to that described by Keynes, but in his case the foamy cells had penetrated into a larger number of fat globules and the diagnosis by frozen sections was difficult. Keynes describes the predominant cell in the lesion as an embryonic fat cell. This appears to be rather confusing, as, although the cells taken singly are morphologically indistinguishable from the cells of developing adipose tissue, they are obviously functioning as fat phagocytes, probably of endothelial origin and accompanied by foreign-body giant cells. The myelin phagocytes or 'Gitterzellen' found in the central nervous system in disease are closely related cells. In this case the early stages in the absorption and invasion of an injured fat globule could be traced from the earliest phase when the fat was breaking up, but was not yet invaded by phagocytic cells, to the later phase when a mass of endothelial phagocytes entirely replaced it. Judging from the other published descriptions, the lesion then undergoes progressive fibrosis at its edge; the central mass of fat phagocytes breaks up, leaving a cyst containing semi-fluid fat, surrounded by a zone of giant cells and walled in by fibrous tissue.

I am much indebted to Mr. Duncan Wood, Surgeon to the Bristol General Hospital, for permission to use his clinical records.

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*SHORT NOTES OF  
RARE OR OBSCURE CASES*

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**A PEDUNCULATED THYROID TUMOUR AT THE BASE OF  
THE TONGUE, ARISING FROM THE REGION OF  
THE FORAMEN CÆCUM.**

By FRANK HARVEY, LONDON.

TUMOURS consisting of thyroid tissue which grow at the base of the tongue are portions of the thyroglossal mass which develops from the hypoblast in the middle of the furrow that lies between the two elevations on the floor of the primitive pharynx, from which the buccal and pharyngeal parts of the

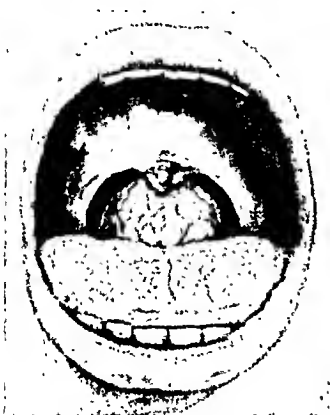


FIG. 486.—Thyroid tumour of the tongue.

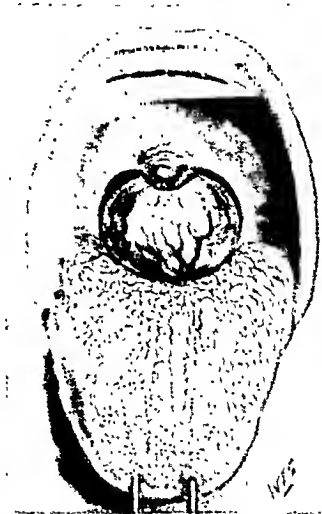


FIG. 487.—Appearance of tumour with tongue held forwards.

tongue are formed.<sup>1</sup> These tumours are more often solid than cystic, consisting of vascular thyroid tissue of immature development. When growing in the region of the foramen cæcum they are generally sessile, and rarely pedunculated as in the case to be described.

**HISTORY.**—On July 2, 1924, I saw with Dr. Harold Greenish, of Fleet, Hants, a girl, age 26 years, who had had a severe hæmorrhage from the mouth on the previous day. The patient had complained to her parents for some months of difficulty in breathing and swallowing; but little

notice was taken of her complaints, owing to her being considered mentally deficient.

For two years thyroid extract had been given with good effect, but had been discontinued, as the patient became very troublesome and refused to take it, and the parents were apathetic. Menstruation commenced at 14 years, and is, at the present time, regular. The patient's parents are normal individuals, as are the other members of the family.

ON EXAMINATION.—The girl is cretinoid, stunted in growth, being only 4 feet 4 inches in height, undeveloped mentally, talks and behaves like a child of eight or nine years of age. Skin and hair are normal. No thyroid gland is palpable.

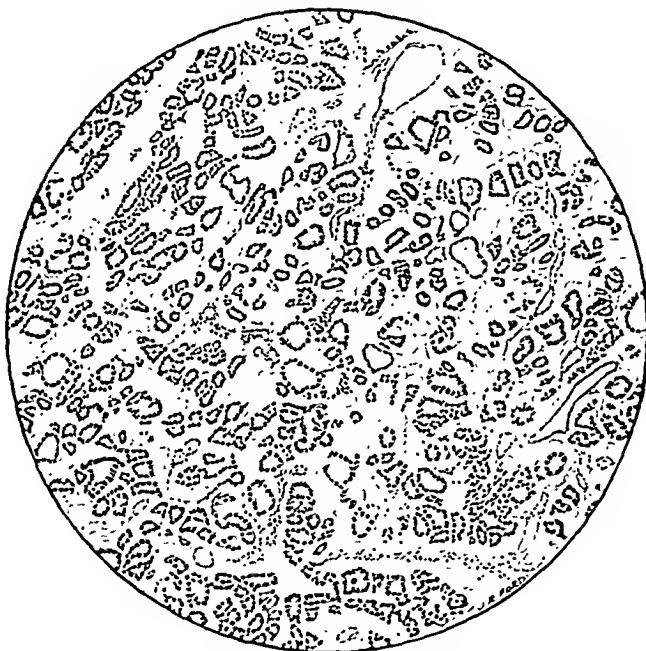


FIG. 488.—Microscopical appearance of a section of the tumour. ( $\times 65$ .)

The patient was anæmic when I first examined her, from the hæmorrhage of the previous day. On looking inside the mouth a tumour could be seen lying at the back of the tongue nearly filling the space between the anterior pillars of the fauces and pushing the uvula forwards (*Fig. 486*). The tumour was mobile, about the size of a walnut, pinkish in colour, and with large blood-vessels coursing over it. By using a small laryngeal mirror it was possible to see the attachment of the tumour to the base of the tongue, which was by a short, thick pedicle, about a quarter of an inch in thickness. The epiglottis could not be seen. As the presence of the tumour was causing dyspnœa and dysphagia, and also because of the severe hæmorrhage that had occurred on the previous day, it was decided to remove it.

OPERATION, July 4, 1924.—The patient was given a general anæsthetic. A Doyen's gag was introduced and the tongue held forwards by a clip (*Fig. 487*). To avoid hæmorrhage two curved needles threaded with No. 2 catgut were passed under the pedicle. An elliptical incision was made with a scalpel and the tumour with its pedicle was removed. The sutures were tied and effectually controlled the hæmorrhage, which was not severe. Convalescence proved uneventful. The patient has taken thyroid extract since the operation eighteen months ago, with marked improvement in her mental condition.

MICROSCOPICAL EXAMINATION (*Fig. 488*).—Dr. Cavendish Fletcher and Dr. H. A. Osborne kindly examined the tumour for me and reported as follows :—

“The specimen is a soft rounded mass about the size of a large walnut. It is dull-pink in colour and one section was found to be mainly solid with a small central lumen which on histological examination proved to be a false cyst due to free hæmorrhage and degeneration—a common occurrence in thyroid tumours. The solid portions of the tumour are composed of a cuboidal epithelium arranged in alveolar and tubular masses—sometimes solid but frequently displaying a central lumen. The stroma is of connective tissue—frequently hyaline—and there is a copious blood-supply. There is no definite colloid. The tumour is encapsuled and obviously an adenoma, resembling a thyroid foetal adenoma.”

I have looked through the literature of thyroid tumours in the region of the tongue, but have not found any reference to one with so definite a pedicle, and Sir James Berry, who has taken a kindly interest in the preparation of this paper, tells me he has never seen one.

### CONCLUSIONS.

1. Islets of thyroid tissue met with along the course of the thyroglossal tract are portions of the thyroglossal mass which develops at the base of the tongue, and are probably left behind as that mass sinks down the neck to occupy the normal position of the thyroid gland.
2. These tumours are generally sessile and rarely pedunculated.
3. In the majority of cases operative procedures are contra-indicated, owing to the danger of inducing myxœdema. It is only when these tumours cause definite symptoms, such as severe dyspnœa and dysphagia, that operative treatment is called for.

Finally, I wish to acknowledge my indebtedness to Mr. Ralph Coyte for correcting the proofs of this paper, to Mr. Thornton Shiells for his beautiful drawings of the case, and Mr. Ford for his drawing of the microscopical section.

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## APPENDIX 'PINNED' TO THE LIVER.

BY WALTER W. GALBRAITH, GLASGOW.

A MALE child, age 11, was admitted to the Royal Hospital for Sick Children, Glasgow, on Dec. 14, 1923. He was stated to have been in his normal health until Dec. 8, when he complained of headache and slight colicky abdominal pain. The pain was not severe, and he was able to go about; but on the next day he vomited once, and, as he still complained of pain, he was confined to bed and given a dose of castor oil, with the result that his bowel moved freely. On the succeeding days the abdominal pain, more severe and almost constant, became localized in the right upper quadrant and was aggravated by deep breathing.

ON ADMISSION.—The patient looked very ill. The face was flushed, the respirations were rapid and shallow, and the *alæ nasi* moving. He stated that the pain was on the right side of the chest and the right upper quadrant of the abdomen, and was so severe that he was frightened to take a deep breath. There was a history of a right-sided pleurisy six months previously, and in infancy he suffered from anterior poliomyelitis affecting both legs.

EXAMINATION.—There was tenderness all over the abdomen, but rigidity was confined to the right hypochondrium, and over that area there was extreme tenderness on pressure. The respiratory murmur was diminished at the base of the right lung, but there were no adventitious sounds or other evidence to suggest an intrathoracic lesion of sufficient gravity to account for his condition. The heart sounds were normal. Temperature 102°, pulse 124, respirations 32. In our opinion the child was suffering from an acute abdominal condition, the most likely cause for which seemed to be inflammation of a high appendix resulting from an incomplete descent of the cæcum.

OPERATION.—Two hours after admission the abdomen was opened by a right rectus incision, and there was at once evident a fair amount of free fluid in the abdominal cavity, some of which was taken for culture. The cæcum was found raised out of the right iliac fossa and slightly pulled upwards by a tight band which lay on the anterior surface of the ascending colon. This band stretched from the normal site of the appendix to the under surface of the right lobe of the liver, to which it was firmly attached. Easily identified as the appendix, it was divided at its proximal end, the stump being ligated and buried in the usual manner. It was then dissected up towards the liver, its mesentery and one or two adhesions being divided. The whole appendix was now free except the tip, which was isolated from the general abdominal cavity by gauze packing and gradually freed by blunt dissection. The adhesion was dense, but it ultimately came away from the liver with a jerk. It was then seen that the difficulty in removing the appendix was due to a pin projecting from its tip and firmly fixed into the liver. There was no evidence of the presence of an abscess in the liver, but a tube was led to the surface from the puncture caused by the pin. The abdomen was closed in layers. After the operation the child recovered somewhat, and the temperature fell to 99°, pulse 108, and respirations 28; but three days later he

had a rigor, and the temperature rose to  $102.8^{\circ}$ , respirations 48, pulse 140. The face was flushed, and there was a faint icteric tinge, the breath being foul and smelling slightly of acetone. There was impairment of the percussion note and of the respiratory murmur at the right base. The general condition of the child was grave, and he died a few hours later.

**POST-MORTEM EXAMINATION.**—At the examination the next morning there was found a well-marked fibrinopurulent exudate throughout the abdominal cavity, the upper surface of the liver being covered with a thick layer. The liver itself was much enlarged, and on section it was found that rather more than the upper half of the right lobe was the seat of multiple

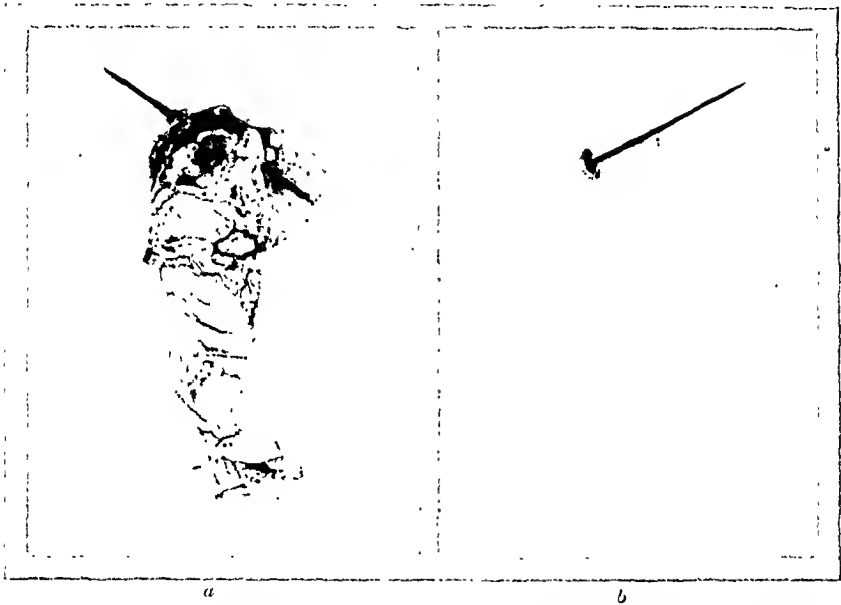


FIG. 489.—*a*, The appendix with the corroded pin projecting from its lumen. *b*, Skiagram showing the calcareous deposit on the part of the pin within the lumen of the appendix.

confluent abscesses, some of which just reached the surface of the organ, and surrounding this area there was a zone of acute inflammation hæmorrhagic in appearance (*Fig. 490*). The remainder of the liver substance showed marked fatty degeneration. The spleen was enlarged and soft, and the kidneys were fatty and showed catarrhal changes. The lungs were acutely congested, and there were numerous petechial hæmorrhages on their surface. There was a diaphragmatic pleurisy on the right side. Fatty degeneration of the heart muscle was noted. No organisms either in film or on culture were found in the free fluid removed from the abdominal cavity at the operation. The appendix, which had been removed at the operation, was partially opened, when it was seen that the head of the pin was surrounded by calcareous deposit, and that there were numerous thread-worms within the lumen. The rest of the pin was corroded (*Fig. 489*).

**Comments.**—Cases of foreign body in the appendix are not rare, but

those with a pin in the appendix are not common. The above case, in which a pin became lodged in the appendix, and made its way to and perforated the tip, which it thereby fixed to the under surface of the liver, causing an abscess, must be one of extreme rarity, if not previously unrecorded. The child never



FIG. 490.—Shows a section of the liver (natural size), with confluent abscesses on the upper surface of the right lobe. The point where the pin was fixed, and its direction, is indicated by an arrow; the track is not seen in this section.

remembered having swallowed a pin, and his mother could throw no light on the matter. The attack of pleurisy six months previously, which according to his doctor was a genuine attack, was probably an early manifestation of the presence of the pin in the liver substance.

## A FIBROMATOUS TUMOUR CONTAINING OSTEOCLAST-LIKE CELLS GROWING IN THE SUBCUTANEOUS TISSUE.

By A. PINEY, London.

THE description of the histology of neoplasms growing in unusual situations is often of value in throwing light on the origin of the growth. I have been unable to find any description of a tumour with the characters of a 'myeloid sarcoma' arising in the skin (Jores,<sup>1</sup> Kaufmann,<sup>2</sup> MacLeod<sup>3</sup>), but the present case seems to show that so surprising an event can occur.

The tumour was removed from the skin of the posterior fold of the axilla of a woman, about 35 years of age. The mass was of about  $\frac{3}{4}$  in. diameter, and had caused both discoloration and puckering of the skin. On section



FIG. 491.—New-formed blood-vessel in the tumour mass. ( $\times 500$ .)

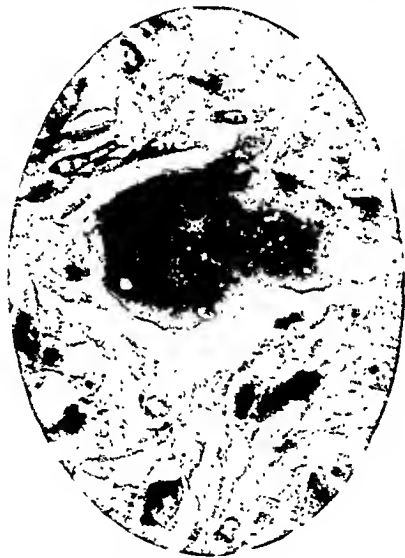


FIG. 492.—Giant cell lying in the midst of fibroblastic stroma. ( $\times 600$ .)

the growth was almost black, and was not sharply demarcated from the surrounding tissues. Some slightly enlarged axillary glands were also removed, but showed no change except the presence of a little ferruginous pigment.

The histological appearances varied somewhat in different parts of the growth, but the most striking feature was the presence of large amounts of dark-brown pigment, which gave the Prussian-blue reaction, i.e., was not melanin. The ground substance of the tumour was composed of active-looking fibrous tissue in which were a number of new-formed vessels with apparently incomplete walls (*Fig. 491*). There were a very large number of giant cells embedded in the fibrous ground substance (*Fig. 492*). These were extremely large elements with many nuclei, but there was no sign either of cell division or of nuclear activity. The giant cells were least numerous in

the places where pigment was most abundant, i.e., they were not collected around the foreign substance. The tissues most densely infiltrated with pigment were, in places, the seat of degeneration which had resulted in cyst formation (Figs. 493, 494). The characters of the tumour were practically identical with those of the 'myeloid sarcoma' of bone.

Professor M. J. Stewart, of Leeds, has kindly examined a section, and writes: "I must say that the giant cells in the central part of the tumour are extraordinarily like those of the benign myeloid

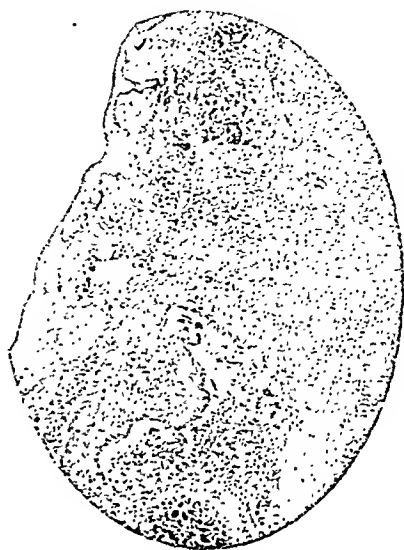


FIG. 493.—General view of the tumour. At the bottom are seen small cysts containing pigment; on the top is the more cellular part of the mass, in which giant cells can just be detected. ( $\times 50$ .)



FIG. 494.—Cyst formation near the skin. ( $\times 150$ .)

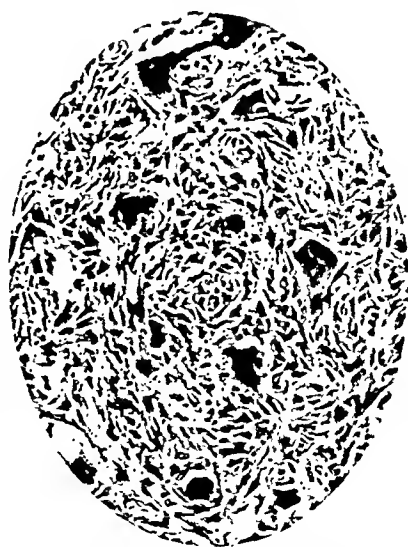


FIG. 495.—General view of that part of the tumour in which giant cells were plentiful. ( $\times 120$ .)

tumour of bone, and not obviously foreign-body giant cells. . . ." He nevertheless says: "I do not think you will find many to agree with you that this should be called a myeloid sarcoma (of bone type)." It must be recalled that Professor Stewart holds that the 'myeloid sarcoma' of bone is a fibrous-tissue tumour derived from a specialized type of fibrous tissue, the peri- or endosteum of bone, which forms osteoclasts. The giant cells of 'myeloid sarcoma' are a continuation of these osteoclasts, which have become definitely neoplastic. He suggests,

in his letter to me, that it is possible that a somewhat similar condition might arise where there is fibrous-tissue reaction together with foreign-body giant cells, e.g., in the skin. He admits that this view would seem to be in favour of the conception of an endothelial origin of the giant cells, but he prefers to presume that they are of fibroblastic origin. If the large cells in the present tumour are really identical with those of 'myeloid sarcoma' of bone it would be essential to suppose that periosteum and endosteum are not very specialized forms of fibrous tissue, and therefore are not specifically able to give rise to this very special type of cell.

Mallory<sup>4</sup> has always supported the view that these tumours in bone represent no very specific type of neoplasm, but that the giant cells are nothing more than special differentiations of endothelial cells.

The very existence of such a tumour in the skin throws light upon the genesis of these neoplasms, which can no longer be so certainly regarded as derivatives of very specialized fibrous tissue.

Professor Mallory writes thus: "My diagnosis is a slowly growing type of capillary hæmangioma. As a result of injury there has been much hæmorrhage with hæmosiderin formation from it, and phagocytosis of the pigment by endothelial leucocytes with formation of numerous foreign-body giant cells (much more common with hæmatoidin crystals than with hæmosiderin granules)".

Parkes Weber has also suggested<sup>5</sup> that the tumour is of histiocytic origin.

I wish to thank Dr. J. M. H. MacLeod for the clinical information, and Mr. C. J. Marshall who removed the growth.

#### REFERENCES.

<sup>1</sup> JORES, *Aschoff's Pathologische Anatomie*, 1923, ii, Leipzig.

<sup>2</sup> KAUFMANN, *Spez. Path. Anat.*, 1923, Leipzig.

<sup>3</sup> MACLEOD, *Diseases of the Skin*, 1920, Lewis, London.

<sup>4</sup> MALLORY, *Principles of Pathologic Histology*, 1914, Saunders.

<sup>5</sup> PARKES WEBER, F., *Proc. Roy. Soc. Med. (Dermat. Sect.)*, 1926, xix, 17.

### SARCOMA OF THE SMALL INTESTINE IN A BOY OF THREE YEARS, ASSOCIATED WITH INTUSSUSCEPTION OF THE ILEUM.

By ANDREW FULLERTON, C.B., C.M.G., BELFAST.

A BOY, age 3 years, was admitted to the Belfast Hospital for Sick Children, on May 15, 1925, with the following history: Up to six days before admission he had been in good health. He then began to suffer from crampy pains in his abdomen, with vomiting and absolute constipation.

ON ADMISSION.—The patient complained of pains all over the abdomen, which was tender on palpation, slightly distended, and hyper-resonant. There was no rigidity, and no tumour could be detected either on abdominal palpation or per rectum. An enema was given with a fair result. The urine was acid, sp. gr. 1020, free from albumin and sugar, but gave the reactions for diacetic acid. Pulse 120, temperature 98°, respirations 30.

I saw the boy on the morning of the 16th. Notwithstanding the fact that an enema had had some effect in clearing his bowels, the abdomen was distended and tympanitic, and the patient continued to vomit. The vomiting was propulsive in character, and occurred immediately after his feeds. There was no faecal odour from the vomited matter.

OPERATION.—On the same day the abdomen was opened by a right paramedian incision below the umbilicus. On opening the peritoneal cavity, free fluid escaped. An entero-enteric intussusception in the lower ileum was found, about a foot in length. This was reduced with comparative ease, disclosing at the apex a growth situated at the mesenteric border of the intestine (*Fig. 496, A*). The gut was distended above the growth and partially collapsed below. The segment containing the growth was removed, and the continuity of the bowel was restored by an end-to-end anastomosis, using two rows of fine catgut suture. On further examination a thickened area was discovered about a foot distal to the tumour, again occupying the mesenteric border. The condition of the child did not permit of further interference. The mesenteric glands were enlarged over a wide area. The abdomen was closed in layers without drainage. The child was discharged on June 6, looking fit and well.

On examination of the excised portion of intestine, the wall at the site of the growth showed a dimple on its peritoneal aspect, surrounded by a hard margin. The gut, on being slit up, exposed a firm swelling about the size of a walnut, projecting into the lumen. The intestinal surface of the growth was sloughy and foul-smelling. On section the tumour was whitish in colour and firm in consistence. On microscopical examination by Professor Symmers it proved to be a round-celled sarcoma.

The patient attended at the out-patient department from his discharge till July 11, when I again examined him. He looked well, but complained of colicky pains in his bowels. On palpation a hard lump could be felt to the right of and below the umbilicus. This was freely movable, rounded in shape, and appeared to be about the size of a small apple. Bimanually, the

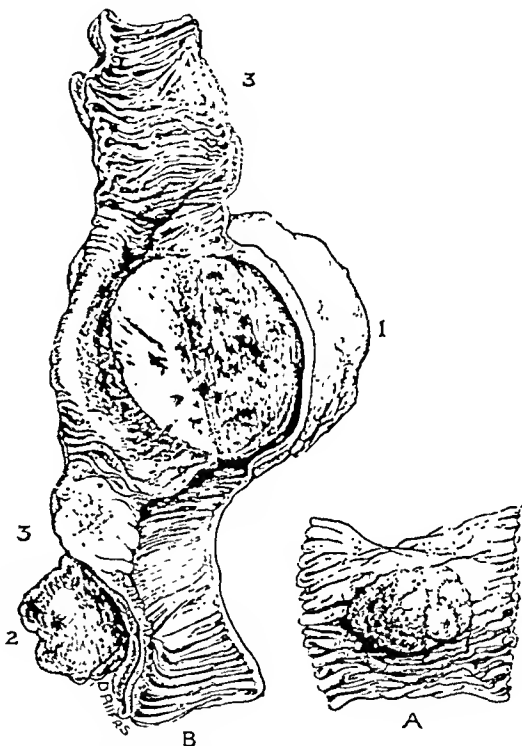


FIG. 496.—A, Segment of bowel removed at first operation, showing growth involving mucous membrane. B, Segment of bowel removed at second operation, showing growths at 1 and 2 and Peyer's patches at 3. The growth at 1 projects well into the lumen of the bowel; that at 2 is in the mesentery at its junction with the bowel.

tumour could be felt and pushed about quite freely in the peritoneal cavity. At the urgent request of his mother, the patient was readmitted to the wards on July 15, though it was felt that any treatment that could be carried out would be only palliative.

**SECOND OPERATION.**—On July 18 the abdomen was again opened, by an incision close to the scar of the previous one. A search was first made for evidence of the former operation on the ileum. The junction was discovered with difficulty, the site of the anastomosis being represented by only a very thin white line. There were no adhesions and no constriction of the gut. About a foot distal to this, and a foot proximal to the ileocaecal junction, a large growth was found (*Fig. 496, B*). This was evidently the thickened area seen at the former operation, which had greatly increased in size during the two months intervening, measuring now  $2\frac{1}{2}$  in. by  $1\frac{1}{2}$  in. It projected into the lumen of the gut from the mesenteric border. The affected segment of bowel was removed and an end-to-end anastomosis carried out. A number of small areas of thickening, evidently new growth, were found, all at the mesenteric border, reaching as far up as the duodenojejunal junction. The mesenteric glands were much enlarged. The vessels of the mesentery, more especially the veins, were engorged. The large intestine, the liver, spleen, stomach, and bladder were examined, but showed no evidence of disease. The abdomen was closed in layers.

The patient had some abdominal distention and a good deal of vomiting after the operation, but was relieved by enemata on the fourth day. His condition then began to improve, and he was discharged in apparent good health on Aug. 4, his only complaint being a feeling of fullness after meals.

**FURTHER PROGRESS.**—Aug. 22. Boy looks the picture of health, and is free from pain. Nothing abnormal can be detected on abdominal examination.

Sept. 25. The child complains of crampy pain all over abdomen, worse after a meal. The patient has lost flesh and colour. The abdomen is distended and tympanitic. Indistinct lumps can be felt through the abdominal wall. Per rectum, two large, firm, rounded masses can be felt, one in front and one on the right side, apparently adherent to the pelvic wall.

Oct. 17. The mother reports that the child is too ill to attend at the Hospital. He has lost much flesh and colour, suffers from thirst, has pains in the lower abdomen, and watery diarrhoea. His feet, legs, and scrotum are swollen.

The patient died at home on Oct. 23. A post-mortem examination was not obtained.

**Comments.**—The points of importance in the case are :—

1. Sarcoma of the small intestine is very rare, especially in children. Corner and Fairbank<sup>1</sup> collected 103 cases from the literature. Of these, 65 (63 per cent) were present in the small intestine, the largest number being in the ileum, and 38 (37 per cent) in the large intestine. The age of greatest frequency in the small intestine was between 30 and 40 years. W. Maxwell Telling<sup>2</sup> describes a case, similar in some respects to the present one, in a boy, age  $3\frac{1}{2}$  years.

2. The situation of the growths in close association with the vessels at the mesenteric border raises the suspicion that they were embolic and



secondary to an unrecognized primary growth elsewhere, possibly in the pelvis, where, later in the case, large tumours were discovered apparently growing from the pelvic bones.

3. The presence of an intussusception of the small intestine, due to the growth.

4. The recovery after enterectomy on two occasions, within two months, in a young child.

5. The complete restoration of the bowel as seen two months after an end-to-end anastomosis.

Although both these operations were quite futile as regards ultimate recovery, they gave temporary relief and prolonged life, and at least satisfied the friends of the patient that everything had been done to save him. The second operation was perhaps hardly worth while, having regard to the state of affairs found at the first.

#### REFERENCES.

- <sup>1</sup> CORNER and FAIRBANK, "Sarcomata of the Alimentary Canal", *Trans. Pathol. Soc.*, 1903, lvi.  
<sup>2</sup> TELLING, W. MAXWELL, *Proc. Roy. Soc. Med.*, 1920, xiii, No. 9.

### HYPERNEPHROMA OF GALL-BLADDER : CHOLECYSTECTOMY.

BY WILSON TYSON, LOWESTOFT.

WITH A REPORT ON THE SECTIONS BY A. PINEY, LONDON.

As there does not appear to be any record of a growth of the gall-bladder presenting the structure regarded as typical of a hypernephroma, the notes of the following case may be of interest.

Mrs. C., age 63, for the last nine months has complained of flatulent dyspepsia, with discomfort and occasional attacks of pain in the right upper abdomen, apparently not sufficiently severe to be typical of gall-stone colic; there has been no jaundice or urinary symptoms.

EXAMINATION.—On Aug. 6 I was asked to see her because of an attack of pain in the upper abdomen. There was a tender, hard swelling to be felt below the right costal margin which was considered to be an enlarged gall-bladder; there was no other abdominal swelling, and no evidence of a tumour in either lumbar region. The urine was free from blood, albumin, pus, or bile. Operation was advised.

OPERATION.—On opening the abdomen by a right upper paramedian incision, the gall-bladder was found to be the seat of a growth somewhat irregular in outline; the liver was smooth, and to the naked eye free from growth; no other abnormality was found in the upper abdomen. The cystic duct where it joined the common bile-duct appeared normal in size; the gall-bladder, which was free from adhesions, was removed, a tube was passed down to the ligated end of the cystic duct, and the abdominal wound closed. No gall-stones were found.

Progress was normal. At the present date (March 19) she is free from pain and is putting on weight.

DESCRIPTION OF THE SPECIMEN.—On section the gall-bladder was a mass of growth; the cystic duct was distended near the gall-bladder, but at its junction with the common duct appeared normal. *Fig. 497* shows the gall-bladder on section, about half size, after hardening. Portions of the growth were sent to the Clinical Research Association, and the report was as follows:—

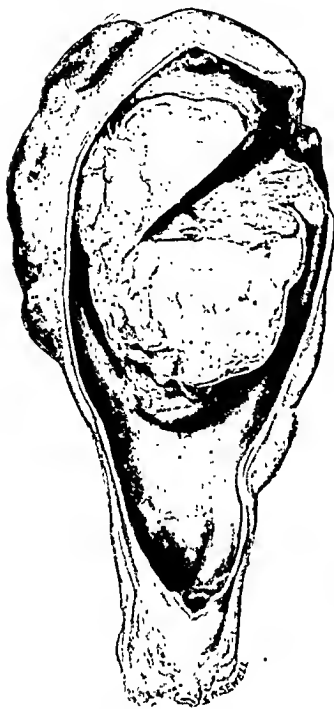


FIG. 497.—Appearance of the gall-bladder on section.

“*Section A*: The section shows blood-clot and eroded tissue surrounding a nodule of malignant new growth which is of the type usually described as a hypernephroma.

“*Section B*: Nearly the whole of this tissue is malignant, showing the peculiar clear cells which are observed in *Section A*. At its margin is a small portion of liver tissue; the parenchymatous cells show fatty degeneration and the surrounding fibrous tissue is increased in amount. A small strip of necrotic tissue at the opposite pole shows bile staining, and evidently indicates the lumen of the gall-bladder.”

I have submitted the sections to Dr. A. Pincy, Director of the Institute of Pathology, Charing Cross Hospital, who has been good enough to have the photomicrograph done and has given me permission to append his comment on the case.

REPORT ON SECTIONS OF ‘HYPERNEPHROMA’ OF THE GALL-BLADDER (A. Pincy).—The histological characters of this tumour are identical with those described as being typical of hypernephroma by Grawitz<sup>1</sup> in 1883. No portion of gall-bladder can be identified in the sections, but at one side there is a small portion of liver. The histological characters of the tumour are seen in *Fig. 498*, and the clear cells with distinct walls supported by a fibrous stroma cannot be confused with those of any other neoplasm.

The present tendency is to regard renal hypernephromata as being derived from altered kidney-cells, and there can be no doubt that true adrenal rests in this



FIG. 498.—Section of ‘hypernephroma’ from gall-bladder. The extremely clear cytoplasm with distinct cell walls is clearly shown, as is also the fine supporting fibrous stroma. ( $\times 150$ .)

organ are far from common, although the theory that hypernephromata grow from such structures would presume their comparatively frequent presence.

Adrenal rests are extremely common in some situations, e.g., along the spermatic cord, in the broad ligament, and on the under surface of the liver, but no record of such a structure in the wall of the gall-bladder is available.

Borst<sup>2</sup> is still strongly of the opinion that the majority, if not all, hypernephromata are of adrenal origin, and there is much to support this view; if it be accepted, the explanation of the genesis of the present tumour would not present insuperable difficulties. The neoplastic growth either of an adrenal rest in the liver near to the gall-bladder or of such a rest in the wall of the latter organ would well account for its occurrence.

## REFERENCES.

<sup>1</sup> GRAWITZ, P., *Virchow's Arch.*, 1883, xciii, 39.

<sup>2</sup> BORST, M., *Aschoff's Pathologische Anatomie*, 1923, i, 755.

## FUSIFORM DILATATION OF THE DUODENUM SIMULATING HOUR-GLASS STOMACH.

By JOHN MORLEY, MANCHESTER.

THE condition of the duodenum encountered at operation in the case recorded below presents such interesting features, and is of such striking rarity, that it is considered worthy of publication.

A married woman, age 61, was referred to me in December, 1924, by Dr. J. A. Lees, of Rawtenstall, on account of gastric trouble. She gave a history of pain in the stomach for the past ten years, at first intermittent but for the past few months continuous. The pain would commence about five minutes after food, and last for several hours. It was so severe that she often applied hot flannels to the abdomen. Some relief was given by alkalis. There had been no vomiting and no hæmatemesis. The appetite had been conspicuously poor for many weeks. Of late she had suffered from alternate constipation and diarrhœa, and during the last six months she had lost weight considerably. An X-ray examination five years before had shown 'an ulcer on the stomach'.

EXAMINATION.—The patient was a thin woman, of sallow complexion, with typical *habitus enteroptoticus*. Examination of the abdomen revealed marked gastroptosis associated with a tender spot in the mid-line just above the umbilicus. Both kidneys were movable.

On clinical grounds the case was regarded as one of gastroptosis associated with general splanchnoptosis, with possibly a gastric ulcer in addition. She was referred to Dr. R. S. Paterson for X-ray examination, and he reported as follows: "The screen examination in this case showed the stomach lying extremely low, the pylorus being at the level of the 5th lumbar vertebra. There is, however, in addition to this, what appears to be an organic hour-

glass condition of the stomach with a small sac at the pyloric end (*Fig. 499*). There was a small filling defect in the constricted area between these two sacs. Examined five hours after an opaque meal, a delay was seen in both sacs. Food could be pushed from one sac into the other. Nothing abnormal was seen in the small intestine. The colon was lying very low, both the hepatic and splenic flexures being well below the line of the iliac crests."

After this examination I advised operation on the ground that, in addition to the enteroptosis, the patient was suffering from hour-glass contraction of the stomach resulting from chronic ulcer.

OPERATION, Jan. 13, 1925.—Under ether anaesthesia an incision was made in the mid-line above the umbilicus. What appeared at first sight to be a

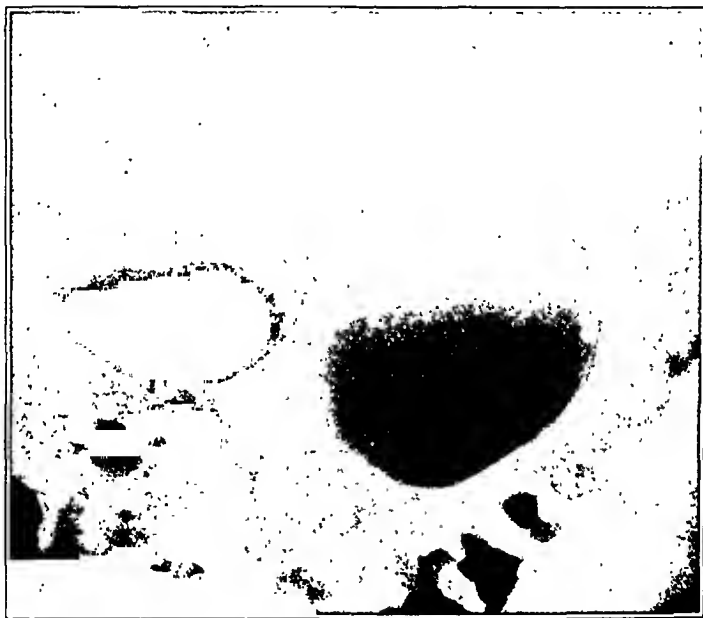


FIG. 499.—Showing barium in stomach (right sac) and dilated first part of duodenum (left sac).

typical hour-glass stomach was disclosed. The healed scar of a small ulcer was found on the lesser curvature about two inches above the constriction between the two sacs. On examining the outlet of the large distal sac, no pyloric ring could be found, but the sac terminated abruptly in the second part of the duodenum, which ran downwards in front of the right kidney. This part of the duodenum was unusually narrow and empty, but otherwise healthy. It was now realized on closer inspection that what appeared to be the distal sac of an hour-glass stomach was actually the first portion of the duodenum, which had undergone an extreme degree of fusiform dilatation. The waist of the apparent hour-glass stomach was formed by the pyloric ring, which showed no pathological change beyond some slight hypertrophy of the circular muscle coat. The narrowing of the second or descending

portion of the duodenum appeared to be due to the pressure of the movable right kidney. The kidney was rather more rotund and firmer than usual, and could be drawn downwards and inwards on the psoas shelf with great ease, when it pressed the second part of the duodenum forward and appeared to obstruct it. It was evident that in the upright position this pressure on the duodenum from behind by the movable kidney must be exerted continuously.

A posterior gastrojejunostomy was performed in the usual manner, and in addition an anterior duodenojejunostomy was performed on the front of the dilated portion of the duodenum. On account of the frail condition of the patient, nephropexy was not considered advisable.

Convalescence from the operation was uneventful. The patient was fitted with a corset-belt for the splanchnoptosis, and four months later reported that her health was improving very satisfactorily.

## IMPLANTATION DERMOID OF THE TERMINAL PHALANX OF THE THUMB.

By HAROLD BURROWS, C.B.E., PORTSMOUTH.

THE patient, an engineer, age 47, was sent to me by Dr. C. M. G. Elliott in February, 1925.

**HISTORY.**—When a boy of about eleven or twelve years he ran a piece of wire deeply into his left thumb. He cannot say whether the wire went into the bone or not; but when the wound healed it left a depression in the skin at the point of entry, and this little crater remained as a permanent mark. He does not remember of what kind the wire was or any other details. The depression left in his thumb was of about  $\frac{1}{16}$  in. diameter and  $\frac{3}{16}$  in. deep, and was situated on the ulnar side of the thumb towards the tip. About twelve months before the present accident he noticed that the end of his left thumb was swollen. Four months previously to my seeing him, a piece of metal had fallen on his left thumb, which had been tender and painful ever since, so that he had been quite incapacitated from work; there was no surface wound, and the nail had not exfoliated.

**ON EXAMINATION.**—When first seen by me the terminal half of the thumb was considerably swollen and reddened; it was hot to the touch, very tender and painful, and the patient said that it was always throbbing. An X-ray photograph taken by Dr. Beverley Bird showed a fibrous irregular vacuolation of the terminal phalanx (*Fig. 500*).

**OPERATION.**—On Feb. 24 I made a lateral incision in order to expose the terminal phalanx, but on cutting through the skin I immediately came down upon a mass of white material



FIG. 500.—Skiagram showing appearance of terminal phalanx of the thumb.

resembling lanolin. It was of a purer white than the sebum found in a sebaceous cyst. This material almost completely surrounded the phalanx, and was not enclosed by any cyst wall. To attempt any palliative measures seemed unpromising, so I removed the terminal phalanx of the thumb, and covered the stump with a palmar flap.

On subsequent examination the phalanx was found to be occupied by a single ramifying cyst which had a white glistening lining, some of which was sent to Dr. Radcliffe for microscopical examination. His report is as follows: "The cyst wall is formed by dense, almost acellular, fibrous tissue, lined by a stratified epidermis which shows a prickle-cell layer and stratum granulosum, but there are no papillæ. It suggests an implantation dermoid."

The operation revealed no sign of pus.

It seems clear that the patient was suffering from an implantation dermoid cyst in the terminal phalanx of his thumb; that the cyst had become ruptured by direct injury, and that the diffusion of its contents among the soft tissues of the thumb had caused a persistent, painful inflammatory reaction leading to disability.

The patient now has a healthy painless stump, and he says that he is hardly incapacitated at all. What he chiefly misses is the thumb-nail.

## REDUCIBLE HERNIA OF A LIPOMA OF THE KNEE-JOINT.

By W. SAMPSON HANDLEY, LONDON.

LILIAN S., age 25, was admitted to the Middlesex Hospital under my care, with a history that nine months previously she had fallen downstairs, bruising the outer side of the left knee, which has since been the seat of pain. On examination a soft semifluctuant rounded swelling was to be felt under the skin opposite the upper edge of the tibia and just on the outer side of the ligamentum patellæ. It was of about  $1\frac{1}{4}$  in. diameter, circular in outline, and flattened in the plane of the skin. Its edge could be felt to slip from under the finger, and the skin was freely movable over it. On its deep aspect the swelling appeared to be fixed. On flexion of the knee the swelling disappeared almost completely, re-appearing suddenly when the joint was again extended. The swelling was thought to be a Morrant Baker's cyst.

On Dec. 8 an incision 2 in. long was made over the swelling, which proved to be a lipoma. It was found to have a pedicle which, entering a small, round, sharply defined hole of  $\frac{1}{8}$  in. diameter in the capsule of the knee-joint, was continuous with a lipomatous mass behind the ligamentum patellæ. Through this opening, in flexion of the joint, the soft lipoma reduced itself into the subsynovial tissue. The opening was enlarged, and a well-defined lipoma was shelled out, the capsule being then sewn up. The patient made an excellent convalescence, and her symptoms were relieved. She was discharged a fortnight after operation. It must remain uncertain whether the injury took any share in causing the abnormal opening in the capsule which permitted the formation of the subcutaneous portion of the lipoma.

## LARGE SALIVARY CALCULUS WHICH HAD UNDERGONE SPONTANEOUS FRACTURE LEADING TO EBURNATION OF THE BROKEN SURFACES.

BY SIR JAMES BERRY, LONDON.

THIS calculus, which I recently removed from a lady, age 46. seems worthy of record, not so much on account of its large size, but because of the peculiar spontaneous fracture which it had undergone long before it was removed.

Twenty-nine years ago, at the age of 17. the patient had first observed a swelling in the right submaxillary region, especially noticeable while she was eating and when she had a cold. For several years she had been aware of a hard lump in the floor of the mouth on the same side. As it gave her but little inconvenience she paid no attention to it until her dentist advised her to see Dr. Hardy Fleetwood, of Hadleigh, who at once passed her on to me.

The calculus was removed without any difficulty through an incision in the floor of the mouth. It was lying at the anterior end of the submaxillary duct. It was then found to have been broken spontaneously into three nearly equal portions. There had been, however, very little displacement of the fragments, which must have been gripped tightly by the duct in which they lay. As the upper illustration shows (Fig. 501), the fragments when fitted together preserved the original shape of the calculus. The very slight amount of movement taking place over a long period of months or years had led to a polishing or eburnation of the opposed surfaces. This condition, common enough in the cases of biliary and prostatic calculi, is, so far as I know, extremely rare in the case of salivary calculi. There was no clinical evidence of the date at which the calculus broke, but it must have been at least many months, or more probably years, before its removal.

The calculus weighed 3.11 grm. Its length was 29 mm. ( $1\frac{1}{8}$  in.) and its greatest diameter 12 mm. ( $\frac{1}{2}$  in.). It is now in the Museum of the Royal College of Surgeons (No. E. 28), which contains no similar specimen showing eburnation of the fractured surfaces of a salivary calculus.

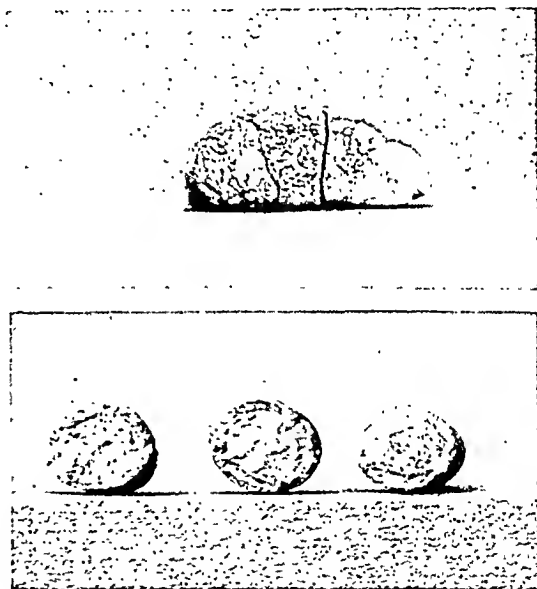


FIG. 501.—Showing lines of fracture of salivary calculus and eburnation of broken surfaces.

## REVIEWS AND NOTICES OF BOOKS.

**Manipulative Surgery: Principles and Practice.** By A. G. TIMBRELL FISHER, M.C., F.R.C.S., Surgeon with charge of Out-patients, Seamen's Hospital, Greenwich. Demy 8vo, pp. 168, with 62 illustrations. 1925. London: H. K. Lewis & Co. Ltd. 7s. 6d. net.

THIS small book represents a laudable endeavour to rescue from mysticism and irregular practice the sound principles of curative manipulation, the value of which has been recognized by such great pioneers as Hunter, Hilton, Paget, and Thomas.

There are three great classes of cases for which manipulation is indicated. These are conditions of adhesion in or round the joints, functional conditions, and subluxations. An accurate and careful account of all the large joints is given, including the special disabilities and the normal range of movements. Greatest attention is paid to the knee, because this joint is so liable to obscure forms of derangement. Probably many of the most successful bone-setters have achieved their results by a combination of shrewd instinct with strong psychic force, and unfortunately neither of these things can be described, much less imparted, by a printed page. Bone-setters appear never to write books or articles descriptive of their methods. They prefer to be written about in the newspapers, and to allow it to be hinted that they are in possession of a profound secret. Mr. Fisher does well to expose the principles of manipulation to light and reason. If in the next edition he could persuade a certain eminent bone-setter and knight to write an appropriate foreword, it would greatly add to the interest of his pages.

**Abdominal Operations.** By SIR BERKELEY MOYNIHAN, Bart., K.C.M.G. In two volumes. Fourth edition, revised. Royal 8vo. Pp. 1217, with 450 illustrations. 1926. London and Philadelphia: W. B. Saunders Co. Ltd. 90s. net per set.

As this book is so well known that it has justifiably reached its fourth edition a set review of it is unnecessary.

No surgical book has yet been written or will be written which is perfect throughout. This, however, is so good and without a rival that we should like to see it better. Those chapters which are the result of the author's vast experience, in which he gets away entirely from the many names that clog others and in which he is dogmatic, could not be better. In some cases symptoms and diagnosis are fully dealt with—for example in acute pancreatitis and in the preliminary observations upon cholelithiasis. In the latter etiology is discussed fairly fully, while it is only very shortly dealt with in the case of gastric ulcer. In the chapter on visceral prolapse we are left without any guidance as to the symptoms and when, if ever, any of the operations mentioned should be performed. The time has come when many of the early references might be omitted; this would provide space to give more fully the opinions of British writers.

We consider this work unrivalled among books on abdominal surgery, and can only express a hope that in future editions we shall have more of the decided opinions of Sir Berkeley Moynihan and more guidance in the border-line cases.

**The Diagnosis and Treatment of Tuberculosis of the Hip.** By G. R. GIRDLESTONE, B.M. (Oxon), F.R.C.S., Hon. Surgeon, Wingfield Orthopaedic Hospital, Oxford. Demy 8vo, pp. 94 + x, with 60 illustrations. 1925. London: Humphrey Milford. 8s. 6d. net.

THIS small book is purely clinical in its scope. An endeavour is made to impress the importance of early diagnosis upon the general practitioner, and to enforce the rule that early suspicious cases should be sent without delay to special institutions



for more detailed examination, instead of allowing time to slip by so that the favourable period for treatment is past. The inclusion of a few cases of pseudo-coxalgia or coxa vara among those sent to hospital does no harm, whereas the postponement of admission of tuberculous hips until bony changes are advanced is a mistake that cannot be remedied. The symptom of limping alone should be enough to indicate rest and further investigation, even if the X rays show no change.

Operation has no place in the treatment of the disease in children, except for the evacuation of an abscess or for the correction of late deformity. Operations for fixation or mobilization are reserved for special cases in adults. The Jones frame is advocated for all cases in the first or active stage of the disease, which is said to be anything from three to twelve months in duration. Details about the fitting and adjustment of the frame are omitted, and we think this is unfortunate, because it is just these details which one looks for in a practical book of this sort. Another aspect, too, we think might be discussed more fully, and that is the methods to be pursued in the case of those patients who cannot receive the ideal sanatorium treatment. The book is excellently illustrated by skiagrams.

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*Pitfalls of Surgery.* By HAROLD BURROWS, C.B.E., M.B., B.S. (Lond.), F.R.C.S., Surgeon, Gosport War Memorial Hospital, etc. Second edition (formerly "Mistakes and Accidents of Surgery"). Demy 8vo. Pp. 325 + x. 1925. London: Baillière, Tindall & Cox. 12s. 6d. net.

THE author and publishers make a strong point of the change of title of this book; but in our opinion the fault does not lie with the title, but with the material it contains. It is such a medley of symptoms, fragments of pathology, and stories of mistakes that we think it is more likely to alarm than to instruct a beginner. One must, however, admit that the motive which has compelled the author to write what is largely a *confessio medici* is supremely altruistic; but many of the dangers and difficulties which are laid stress on seem to us those of twenty-five years ago rather than of to-day, when much experience of so many operators, and a wider knowledge of the pathology of the living, has rid us of many old-time bogies. In fact, one must realize that surgery is too large a subject to be dealt with in a volume such as this, and there are few authors who under the circumstances, dealing with a single aspect of the subject, could avoid being superficial; so that it cannot be urged as a great failing in Mr. Burrows, if, with the best motives in the world, he has made no serious addition to surgical literature.

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*Hand Atlas of Clinical Anatomy.* By A. C. EYLESMEYER, B.S., Ph.D., M.D., Dean of the College of Medicine and Director of Department of Anatomy, College of Medicine, University of Illinois; and TOM JONES, B.F.A., Director of Anatomical Illustration and Instructor in Anatomy, College of Medicine, University of Illinois; with special dissections by O. E. NADEAU, B.S., M.D., Associate in Surgery, University of Illinois. Royal 8vo. Pp. 420 + viii, with 395 illustrations, mostly in colour. 1925. London: Henry Kimpton. 50s. net.

THE best atlas for the study of anatomy is undoubtedly the human body; but in the absence of actual dissections and of the second-best alternative, a large atlas, a hand atlas such as this serves an exceedingly good purpose, though it will be at once admitted that it is impossible to portray any detail of the dissection of the chest or abdomen in a drawing 5 in. by 7 in. The subjects for the drawings and diagrams are well chosen, and, if not particularly selected with a view to their surgical importance, yet there are few which would not be of value to a surgeon. We think the explanatory index of eighty pages a mistake, and should have preferred that the space had been allotted to further diagrams and drawings: the value of such an atlas to a student of anatomy is to supply him with a pictorial illustration of the structures he is reading of in his anatomical text-book, and therefore, in our opinion, such an index is unnecessary.

**St. Bartholomew's Hospital Reports.** Vol. LVIII. Demy 8vo. Pp. 113 + xx. 1925. London: John Murray. 21s. net.

THIS volume contains obituary notices of Klein, Tooth, and Williamson, all written with sympathy and discretion. An article by C. L. Hewer on splanchnic analgesia gives a valuable record of nineteen cases; it is claimed that the shock of large abdominal operations was diminished, but in every case the patient was kept under very light general anaesthesia. Mr. Rawling contributes a very candid description of a case of trigeminal neuralgia which is full of both humour and precepts for those who would learn therefrom.

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**Scoliosis: Rotary Lateral Curvature of the Spine.** By SAMUEL KLEINBERG, M.D., F.A.C.S., Assistant Surgeon, New York Hospital for Ruptured and Crippled: Chief of Orthopædic Service, Israel Zion Hospital of Brooklyn, etc. Medium 8vo. Pp. 311 + xvi, with 140 illustrations. 1926. New York: Paul B. Hoeber, Inc. \$6 net.

THE voluminous literature on scoliosis has tended to enshroud this common deformity in mystery. The author of this monograph has endeavoured to present the subject in a simple fashion, and it must be admitted that he has achieved his object.

In the opening chapters the relation of lateral curvature to the general body mechanism is well emphasized, and particularly its influence on the psychology of the deformed patient. It would be interesting to know whether there is any diminution in the incidence of this deformity as seen in special hospital out-patient departments in the United States. In Great Britain there seems to be no doubt that scoliosis is on the decrease.

In any work on lateral curvature the section on etiology is always apt to be very disappointing to the reader. Dr. Kleinberg gives a brief review of the accepted theories, and with others is compelled to put the incidence of the so-called idiopathic type as 85 per cent. In this group of cases, however, a radiological study of the spine carried out by Buchman, working in the same clinic as the author, has revealed the very frequent existence of osteochondral changes in the epiphyses of the vertebral bodies. The exact significance of such changes cannot be defined at present.

The symmetrical developmental exercises described in the chapter on gymnastic treatment appear rather old-fashioned from the British point of view. The asymmetrical corrective exercises, however, are dealt with more effectively, and should prove a useful guide to the uninitiated.

No extravagant claims are made for the results of forcible correction of fixed scoliosis, but a judicious account of the various methods is presented. The author favours the application of the corrective jacket with the patient in suspension, and from a considerable experience of this method believes that 55 per cent of the cases show definite improvement.

A good account is given of the operative treatment of scoliosis which has been tried fairly extensively during recent years in the clinic of Royal Whitman, work in which the author has collaborated. The technique followed has been spinal fusion over a fair-sized area with the use of a beef-bone graft. In a limited group of cases the results obtained have been quite satisfactory, but the method is still in the experimental stage.

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**Surgery of the Stomach and Intestines.** By J. SHELTON HORSLEY, M.D., Attending Surgeon, St. Elizabeth's Hospital, Richmond. Surgical Monographs, under the editorial supervision of Dean Lewis, A.B., M.D., Professor of Surgery, Johns Hopkins University; Eugene H. Pool, A.B., M.D., Attending Surgeon, New York Hospital; and Arthur W. Elting, A.B., M.D., Professor of Surgery, Albany Medical College. Imperial 8vo. Pp. 325 + xvi, illustrated. 1926. New York and London: D. Appleton & Co. 21s. net.

MANY books have been published on the surgery of the stomach and intestines, but this volume takes the unusual course of treating these subjects from a physiological aspect. The author says in his preface that the large amount of space devoted to physiology requires some explanation. In our opinion it certainly needs

no apology. Physiology is the science of function, and the true ideals of an operation are to remove or correct pathological conditions and to restore as far as possible the physiological functions. How can we do this without a knowledge of the normal workings of the stomach and intestines? Heretofore anatomy has had an almost tyrannical influence over surgery, and we welcome a book which gives to physiology a position of at least equal prominence, and especially when written by so able an author.

The work begins with a chapter on the embryology, and this is followed by another on the anatomy, of the stomach and intestines; there is nothing new in these. Chapter 3 deals with physiology; here the points relative to the proper surgical manœuvres required for the varying pathological conditions are emphasized. The work of many researchers is reviewed, especially that of Alvarez, who by ingenious devices has advanced theories on peristalsis which, though at variance with those of many others, seem to explain clinical phenomena more satisfactorily.

Chapter 4 deals with diagnosis. This chapter is contributed by Dr. W. Higgins, Attending Physician to St. Elizabeth's Hospital, Richmond. He states that the modern conception of gastro-intestinal physiology has brought about radical changes in our interpretations of symptoms arising from these organs. We are indebted chiefly to Carlson and Alvarez for demonstrating the fallacies and unsupported dogmas of former views on this subject.

In Chapter 5 general considerations are dealt with relating to the operating room, anæsthesia, frozen sections, shock and hæmorrhage, and blood transfusion.

Chapters 6 to 15 inclusive are devoted to specific lesions and the appropriate treatment. The author states that an effort has been made to base operations and procedures as far as possible on biological principles, and only those which seem to have such a foundation have been adopted. For example, the work of Dragstedt is very suggestive in showing that it is important not to catch the pyloric portion of the gastric mucosa in the grasp of the suture if it can be avoided. He has demonstrated that one of the most satisfactory methods of establishing an experimental peptic ulcer in the pyloric portion of the stomach of a dog is to insert sutures in the mucosa at one given spot. Towards the cardiac side ulcers are more difficult to create. The author is averse from the use of clamps whilst making an anastomosis, and to ensure a complete mucous covering to the line does not remove any mucosa, and carefully everts this coat, taking special precautions to approximate only and not to crush it by overtightening the sutures, as this would cause sloughing. He uses a lock-stitch very extensively. These points are emphasized with the object of preventing jejunal ulcer, and though we believe these ulcers, following gastro-enterostomy, are more frequent than the 2 per cent usually stated, we cannot agree with Lewishon, whom the author cites, when he says that fully one-third of the gastro-enterostomies done with the usually accepted technique are followed by jejunal ulcer.

In Chapter 16 we have a résumé of the more important research conducted on intestinal obstruction, with especial emphasis laid upon the recent work of Foster and Hausler, and of Haden and Orr. The work of the latter two, dealing with the loss of chlorides of the blood in high intestinal obstruction, promises to have far-reaching effects upon the treatment of this condition. The book terminates with a short chapter on special causes of intestinal obstruction.

A brief bibliography is placed at the end of each chapter. The author says that it is not intended to make this a complete bibliography; only the principal articles or books consulted are given. It seems profitless, he adds, to compile an unnecessarily long list when in these days of accumulative indexes all references can be easily found.

The book is one to be thoroughly recommended. The emphasis given to the physiological aspect of surgery is, in our opinion, of the greatest importance, and the surgery of the future will undoubtedly be much advanced and put on a sounder footing by paying more attention to physiological principles. In this respect orthopædic surgery is a long way ahead of general surgery; but, as this book shows, there are many principles of physiology which have a very direct bearing on the surgery of the stomach and intestines, but are not as yet put into general practice.

**Fractures of the Humerus, Radius, and Ulna.** By ELDRIDGE L. ELLISON, A.B., M.D., Sc.D., F.A.C.S., Professor of Clinical Surgery in the University of Pennsylvania Medical School, with the collaboration of RALPH GOLDSMITH, M.D., and EUGENE P. PENDERGRASS, M.D., both of the University of Pennsylvania. Imperial 8vo. Illustrated. 1925. New York and London: D. Appleton & Co. 21s. net.

THIS volume, one of a series of surgical monographs edited by Drs. Dean Lewis, Pool, and Elting, will prove of special value to those interested in the statistical aspect of fractures, but in many other aspects it is full of interest. The best feature of the volume is the illustrations, which are well chosen and well executed, even if some of the X-ray productions are too small to be clear. The letterpress, which is in a sense subordinate to the illustrations, loses something of its grasp on the memory of a reader by a tendency to excessive classification and production of lists, but everything that is written is clearly founded on personal clinical experience; particularly are the authors definite whenever possible on the question of causation, and one notes with joy that many of the fairy tales which often appeared in surgical writing on fractures from our youth up obtain no place in this volume.

The chapters that have pleased the reviewer most are those dealing with the injuries in the region of the elbow-joint, but the authors do not give due importance to the possibility of secondary Volkmann's contracture, and say little about its prevention; incidentally it is difficult to agree with their statement that it produces a claw-hand, in which we have always been taught the metacarpophalangeal joints are extended, a condition typical of paralysis of the ulnar and median nerves. There are several points in treatment not in agreement with habit in this country, of which perhaps the most marked is the suggestion that the right line of treatment for a fractured olecranon is by splinting, and exceptionally by operation. Again, the authors advise treatment of certain fractures of the forearm in the thumb-up position, a practice which has fortunately disappeared in this country; yet they realize, on p. 273, that supination will be the motion most limited, and in the very next paragraph to that advocating the above-mentioned, and in our opinion misguided, advice, there is a remark so full of truth and of such great value for treatment of fractures in any part of the body that it might be written as a text at the head of each page in such a book as this: "Do not be bound by any rule with regard to . . . position. Dress it in that position and with that fixation that holds it best as shown by the fluoroscope"; with large-minded progressive views like this there is no doubt that the authors in a future edition will have material which will enable them to strike out the paragraph to which exception has been taken.

The last chapter deals with operative treatment, and is altogether on a lower plane of excellence than the rest of the book; for instance, no mention is made of the value of operative treatment when an articular extremity of a bone is involved. There are many cases depicted in this book of fractures similar to this; yet in two years only ten simple fractures have been treated by them by open operation. It is hoped that in the near future wider experience of the value of open operation, combined with the perfected use of an internal splint, such as the Lane plate, will be sought, when those surgeons who have as great opportunities of doing good work as have the workers in this fracture clinic will not any longer hold the opinion that "internal splint fixation is nothing more than the flimsy make-shift at the best, and that absorption soon loosens wires and screws".

**L'Anatomie en Poche.** By Dr. VICTOR PAUCHET and S. DUPRET. Crown 8vo. Pp. 316, with 297 illustrations. 1925. Paris: Gaston Doin et Cie. Fr. 25.

DR. PAUCHET, whilst teaching anatomy and operative surgery at Amiens, designed this small work, and Dupret has carried out his ideas by a series of excellent diagrams. Each page, measuring 7 in. by 4½ in., is occupied by a clear, well-drawn diagram in black and white or in one colour. The drawing is surrounded by the names of all the structures shown. The whole human anatomy is thus dealt with in a small compass, and a most serviceable method is provided of refreshing the memory about intricate facts and names. We hope that this or a similar book may be provided for English readers.

**Rejuvenation by Grafting.** By Dr. SERGE VONONOFF, Director of the Department of Experimental Surgery of the Collège de France, etc. Translation edited by Fred. F. Imianitoff, B.A. Demy 8vo. Pp. 221, with 38 illustrations. 1923. London: Geo. Allen & Unwin Ltd. 15s. net.

THE author, in his introduction, says that between June, 1920, and October, 1923, he performed 52 testicular grafting operations, of which 43 grafts, all transferred from ape to man, form the basis of the observations set forth in the book. The ages of the subjects upon whom the grafts were made ranged from 22 to 75 years. The opening chapter recounts the development of the idea ensuing from observation of the physical and mental characteristics of eunuchs seen in Egypt. The effect of the internal secretion of the testes upon the organism is fully discussed. An interesting account is given of the earliest experiments, which were carried out upon old and useless rams by grafting portions of the testes of young rams into their scrotums. A photograph of an old ram before grafting is shown, and another of the same ram five years after the grafting had been performed. The difference in the appearance of the animal is very pronounced. In order to make sure that the transformation thus obtained was not due to the stimulation of the ram's testicles by the presence of the grafts, the author carried out a further series of experiments in which young goats were castrated and grafted at the same time. The subjects of these experiments preserved their normal appearance and development, and also retained the sexual appetite. The author then goes on to say that the experiments of grafting portions of testes from one animal to another of the same species were so successful that he conceived the idea of experimenting on the human species. As it was impossible to obtain healthy human testes for the purpose of making grafts, he decided to make use of the testes of the anthropoid apes. He claims that the results of these experiments fully justify the operation in those individuals who are the subjects of testicular insufficiency. He asserts that increase in physical energy and improvement in mental capacity are marked, but insists strongly that the introduction of a testicular graft does not act as an aphrodisiac. The details of the operation are carefully described, as also are the means adopted in the preparation of the grafts.

The histories of 44 instances in which the operation was carried out in the human subject make interesting reading, and although in many cases marked improvement in physical and mental vigour became apparent for a time, the effect does not appear to have been lasting, and consequently one is left in doubt as to whether the game is really worth the candle.

**Chirurgie der Sportunfälle.** By Dr. FELIX MANDL, Assistant in the Second University Surgical Clinic, Vienna. Royal 8vo. Pp. 303, with 71 illustrations. 1925. Berlin: Urban & Schwarzenberg. M. 12.

THE author remarks on the rapid extension of sport in late years, especially after the war, and tells us that there are no fewer than 260 football clubs in and around Vienna, with about 60,000 active members. At first sight it does not seem very reasonable to make a special treatise dealing with accidents which arise in the course of sport; but an examination of these pages makes one realize that the author has more than justified his work. He begins with a general description of the type of injuries and the problems of treatment met with in those engaged in active sports, and then devotes a special chapter to the treatment of fractures of the extremities. Valuable suggestions are made for the improvisation of splints from Kramer's metal ladders; these, consisting of longitudinal wires with cross-struts, can be cut and bent for many purposes. There is a lamentable absence of all mention of the Thomas arm or leg splints, which we know so well to be of invaluable service in emergency work. The body of the book consists of a description of the injuries particularly likely to be associated with the different forms of sport. Football, hockey, jumping and running, tennis, duelling, boxing, swimming, riding, Alpine sports, motoring, and flying are among the sports considered, and in each section many facts of interest and importance are collected. We think that the value of the book will be increased if more attention is paid to special methods of 'first aid' which may be useful in emergencies.

**Surgical Pathology.** By WILLIAM BOYD, M.D., M.R.C.P. Ed., F.R.C.S., Professor of Pathology, University of Manitoba; Pathologist to the Winnipeg General Hospital, Winnipeg, 8vo. Pp. 837, with 349 illustrations. 1925. London and Philadelphia: W. B. Saunders Co. Ltd. 45s. net.

THE advent of this book fills a gap which has existed for years in a surgeon's library. To the reviewer the volume has been a constant source of enjoyment, and has been dipped into on innumerable occasions either for information on some particular pathological point, or out of curiosity to ascertain what the author's view might be on some question of examinational interest; the results of these inquiries have usually been inspiring and have never been disappointing.

Hitherto a volume on surgical pathology has rigidly excluded bacteriology, and an article on surgical bacteriology in a text-book usually deals with the subject from so specialized a viewpoint as to be unintelligible to the ordinary surgeon. Here we have bacteriology rightly recognized as a part of general pathology, and written up only to such a level as the surgeon requires and can understand. If one were to criticize the book it would be for the omission of much general pathological chemistry, such as calcium metabolism, the importance of variation in blood-sugar, and the general interdependence of the endocrine glands; these should be no less essential to an understanding of general pathological principles than, for instance, the chemistry of utility of thyroid secretions, on which is written one of the most instructive chapters of the book.

The volume is easy to read and convincing in tone, acquiring the latter quality because every page stamps upon one the conviction that it is written purely upon the author's experience, with no reference to or regard for what may have been the opinion of others or has appeared in other text-books. The author shows evidence of being well read in many matters which are strictly outside that of pure pathology, and his references are extremely well chosen and in every case modern. The illustrations are quite well selected, for typical common conditions are reproduced rather than those of extreme rarity; almost the sole exception is the drawing of anthrax pustules, in which the situation is not typical and the appearance by no means clear. The microphotographs are extremely well reproduced, and the general turn-out of the book is worthy of the high reputation of the publishers.

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**Traitement des Epitheliomas du Maxillaire supérieur par l'Association Chirurgie-Curiethérapie.** By Dr. G. VERCER, Ancien Interne des Hôpitaux de Paris. Royal 8vo. Pp. 204, illustrated. 1925. Paris: Gaston Doin et Cie. Fr. 22.

THE book under review gives an excellent summary of the treatment of epitheliomata of the superior maxilla. The subject is considered from many aspects, and nothing of any importance is left out of the survey.

The author states that, considering the inefficacy of surgical treatment, it was natural to appeal to radiations in the struggle against cancer of the upper jaw. "It is the history of this endeavour as carried out at the Radium Institute of Paris, which we are about to retrace, and of which we are anxious to show the successive steps." They succeeded admirably in the endeavour, and give an instructive résumé of the steps which led to the attainment of the ultimate technique. The achievement of the results detailed is an admirable tribute to Professor Regaud and his colleagues at the Radium Institute. The use of radium as an auxiliary to surgical procedures is clearly shown, and the value of this agent established on a sound foundation. The difficulties of radium therapy are dealt with. Of these, reference is made to two of capital importance: (1) infection, (2) bone necrosis following upon radium exposures. Two other complications are referred to which are peculiar to widespread cancer; they are hæmorrhages and glandular involvement.

In regard to deeply-seated infective processes, the difficulties are described in detail, and the conclusion arrived at is of great importance for the guidance of other workers in the treatment of cancer in these regions. "The only therapeutic agent for this condition is surgical interference on a large scale, free drainage of septic pockets to enable the radiations to act upon tissues relatively free from infection."

Radium bone necrosis is dealt with thoroughly. Much attention is given to the preparation of the site prior to the radium application, and the technique used by Hautart and Monod for packing the radium tubes in the region of the growth.

As regards dosage and quantity, the result of successive treatments with different doses has been to establish what quantity of radiation was necessary to obtain sterilization of the cancer without setting up a necrosis. The actual dose employed is one from 23 to 25 microcuries destroyed per hour in four days; but more important still than the question of the total dose is that of the number of applicators. The same dose concentrated in a small number of applicators will have a disastrous local effect, and practically none on the distant parts; whereas if many applicators are used over a large area, the result is much more satisfactory, the actual areas receiving a dose sufficiently intense to bring about reparative action. The conclusion is reached that the best dose is about 20 to 25 m.e.d. in four days. The quantity of radium to give this dose is placed in 11 to 12 tubes (the actual quantity in each is not stated, but it may be assumed that it is about 50 mgrm. of radium element).

By technique of this kind encouraging results are obtained, those in which the more recent technique has been employed giving relatively better results than in the earlier series. The work described in this admirable treatise is of the highest importance, serving to show that in the struggle against malignant disease it is only possible to obtain satisfactory results by the employment of a technique in which the highest surgical skill is available for collaboration with that of the radium expert.

We have nothing but admiration for the method in which the work has been done, and for the very honest statements of the author. Surgeons and radiologists may well ponder over the contents of this book; they are bound to receive instruction and encouragement from its perusal.

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**Die Chirurgie: A System of Surgery.** Edited by Professor KIRSCHNER (Königsberg), and O. NOROMANN (Berlin). Imperial 8vo. 1925. Berlin: Urban and Schwarzenberg. Fasc. 3, Vol. VI, pp. 228, with 60 illustrations and 10 coloured plates. M. 12; Fasc. 4, Vol. I, pp. 241, with 220 illustrations, M. 12; Fasc. 3, Vol. VI, pp. 321, with 108 illustrations and 4 coloured plates, M. 27.

FASCICULUS 3 forms a monograph on the liver and the pancreas. Professor Heller, of Leipzig, deals with the subject of the liver and biliary system. The sections on the embryology and anatomy are clear and well illustrated, whilst that about hepatic abscess is distinctly meagre, and out of proportion to the rest of the work. Theories of the etiology of gall-stones are dealt with at great length, and the subject is well illustrated, but it is strange that Rovsing's work is not mentioned at all. The X rays with regard to the diagnosis of gall-stones are rather inadequately described, and no mention is made of the bromine or iodine preparations of phenolphthalein in relation to this subject. The various operations and the choice of operative treatment for gall-stones and their complications are well and clearly described. The long list of references to literature contains no reference to Rovsing or Moynihan, and the latter is spoken of in the text as an American surgeon.

Professor Riese, of Berlin, writes about the pancreas, and he gives a good account of the anatomy, pathology, and surgery of this organ. The coloured figures in this section are very beautiful, but their interpretation is sometimes obscure. There is a good discussion on the etiology of acute pancreatitis, in which bacterial invasion and biliary infection both play a part.

FASCICULUS 4 deals with a general study of injuries and wounds and their pathology and treatment by Professor Lotsch, of Magdeburg, and a description of artificial limbs by Dr. Böhm, of Berlin. The section on wounds is very clearly written, and is well illustrated by good microscopical and diagrammatic figures. The description of amputations and excisions is on conventional lines, and possibly greater emphasis might have been laid upon those few amputations which have been proved of service, rather than giving so many of the old classical types. Kinemasthetic amputations are discussed very briefly, and undue prominence is given to a difficult and useless bifurcation operation through the forearm. The section on artificial limbs calls for no special comment. The mechanical details of the upper limbs are unnecessarily complicated for a surgical treatise.

Fasciculus 5 treats of the affections of the kidneys, ureters, urethra, and testes. It is written by Professor Fraughenheim and Dr. Welmer, of Cologne. This forms a very full description of the surgery of the regions concerned, and it shows much more appreciation of foreign work, especially that of Albarran, than is the case in other parts of the system hitherto reviewed. There is a lengthy account of different methods of estimating renal function, but from a practical point of view we think that not enough emphasis is laid on simple methods. Ambard's constant and cryoseopy, both of blood and urine, we think might now well be forgotten, whereas more attention should be paid to the proportion and variation of the nitrogen constituents of the blood and urine. Dealing with movable kidney, the author supports the view that hysteria and other nervous instability form absolute contra-indications to operation. Some rather elaborate and fanciful methods of slinging the kidney to the last rib are described. Nephritis is discussed under a number of headings and varieties, and much attention is given to decapsulation, which seems to have a much greater vogue on the Continent than in this country. The sections on stone in the ureter are rather meagre and poorly illustrated. An ingenious method of plastic reconstruction of the urethra by Budde is illustrated, but otherwise the chapters dealing with the urethra, testes, and penis are on conventional lines calling for little comment. A long account is given of the operative procedures for undescended testes, without adequate consideration of the functional value or after-results of the procedure. This fasciculus is rather sparsely illustrated, and the figures seem to be very unevenly distributed, e.g., there are no fewer than twenty-three figures of operation for phimosis, but not a single illustration of new growths of the testes.

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**Schistosomiasis vel Bilharziasis.** By C. G. KAY SHARP, M.D., Fellow of the Royal Institute of Public Health; Chief Medical Officer, Education Department, Province of Natal. With a foreword by Dr. J. B. CHRISTOPHERSON, C.M.G., M.D., F.R.C.P., F.R.C.S. Crown 8vo. Pp. 74 + v, illustrated. 1925. London: John Bale, Sons and Danielsson Ltd. 7s. 6d. net.

THIS little book is written mainly for the benefit of School Medical Officers and Educationists with a view to drawing their attention to the extent of the disease in Natal, its method of spread, and the means which should be adopted to control it. In the foreword Dr. Christopherson very rightly considers that the best ways of dealing with the problem are: (1) Mass treatment by antimony tartrate in schools and villages; and (2) Educational propaganda to check infection and re-infection.

A brief review of the history and nomenclature is followed by a chapter on etiology. In this the writer points out that in Natal urinary schistosomiasis is the commonest form, and that quite an appreciable number of European school children are affected. A useful map of Natal and Zululand is supplied, showing that, though most of the infected areas are near the coast, there are some from 100 to 150 miles inland. The intermediary hosts in Natal are the snails *Physopsis africana* and *Limnæa natalensis*. This chapter contains an excellent résumé of present-day knowledge, together with several useful illustrations and diagrams.

An account of the pathology and morbid anatomy is followed by paragraphs on immunity and incidence. The latter is perhaps one of the most useful sections in the book, for it contains a record of the writer's investigations, showing that in Durban, with 5000 male school children, 2.4 per cent were known to be infected, while in some native schools in other towns the infection was as high as 8 and 13 per cent.

In the chapter on diagnosis the writer describes his method of examining the urine for ova, which should be very helpful to practitioners. In suspected cases where examination has proved negative he points out that ova may sometimes be dislodged and found in the urine after the passage of a sound.

The recommendations for the prevention of the spread of the disease set forth in the section of prophylaxis are obviously based on considerable local knowledge, and should be very helpful to an M.O.H. in an infested district.

The final chapter contains a review of the treatment of bilharziasis in different parts of the world, but unfortunately does not include details of a scheme of treatment which the general practitioner could easily grasp and carry out. The book concludes with a long list of references to all important work on the subject.





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# ATLAS OF PATHOLOGICAL ANATOMY

ISSUED UNDER THE DIRECTION OF THE EDITORIAL COMMITTEE OF  
*The British Journal of Surgery.*

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FASCICULUS I.

TUMOURS OF BONE.

Compiled by E. K. MARTIN, M.S., F.R.C.S.

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# ATLAS OF PATHOLOGICAL ANATOMY

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## INTRODUCTION



THE study of Morbid Anatomy has of recent years been neglected and has been overshadowed by the advance of bacteriology.

Many surgeons are of the opinion that this is to the disadvantage of the science and art of Surgery, and consider that, in the interests of practical surgery, efforts should be made to stimulate the study of specimens obtained by operation or by autopsy. It is as a result of this feeling that the new *ATLAS OF PATHOLOGICAL ANATOMY* has originated, and it is hoped that many readers of the *British Journal of Surgery* will be gratified to find that the recent great advances in illustrative processes have enabled the Publishers to provide for a much needed want.

It is not to be supposed that any illustration, however good, can take the place of the actual handling and inspection of recent specimens, but it is hoped that the study of typical illustrations of disease will both stimulate and assist in such examinations. In recent years it has been the custom to rely so much upon a report from a skilled microscopist that the information to be gained by a naked-eye inspection at the time of an operation has been often completely neglected. The facilities provided at most large hospitals and for many private patients cannot, however, be always supplied in surgical practice, and it is essential that all operating surgeons should possess sufficient knowledge of morbid anatomy to form a responsible opinion in most cases, and to act upon it.

It is fortunate that the supply of suitable specimens for illustration is practically inexhaustible, and in the Hunterian Museum of the Royal College of Surgeons alone there is enough material for illustrations for many years,

whilst a vast supply is also to be found in the museums of the Medical Schools of London and the Provinces.

In the Hunterian Museum immense changes have been made of recent years, and a great deal is owed to the late Mr. Shattoek. Under his supervision a section of General Pathology has been formed which it is believed is unequalled in the world, and, owing to a new design in the metal and glass cases, the specimens can now be inspected in a better way than ever before.

It will be seen that the majority of the illustrations in the present fasciculus are taken from specimens in this collection, and it will be evident that it is not the intention of those who are responsible for the selection of subjects to depict only rare conditions or diseases. On the contrary, it is of much greater importance that typical examples of the well-recognized diseases should be depicted, for, if the Atlas is to be of practical utility to surgeons, it is certain that it will better serve its purpose by dealing with conditions likely to be encountered in general surgical practice than by illustrating diseases which are very unlikely to be met with. Finally, it is to be hoped that the Atlas will not only be interesting in itself, but that it will also induce its readers to visit the Hunterian and other museums and become acquainted with those specimens which are too numerous to be depicted in any publication.

ANTHONY A. BOWLBY.

## I. SARCOMA OF LONG BONES.

SARCOMA of bone is a malignant tumour composed of cells which, with their accompanying blood-vessels, present the general characters of embryonic connective tissue.

Association with precedent injury is often claimed, but has rarely been established. Pre-existent bone disease appears to be conducive to the development of sarcoma in the case of osteitis deformans, and, among simple tumours of bone, chondroma has a definite tendency to undergo sarcomatous change usually after the lapse of many years.

While sarcoma may commence in any part of a long bone, it affects particularly the extremities, though it has but little tendency to invade the neighbouring joint, both articular cartilage and synovial membrane offering a definite resistance to its passage. The tibia, femur, and humerus are the bones most often affected, and it is at the more actively growing ends of these, i.e., at shoulder and knee, that sarcoma is chiefly found.

The tumour may arise either from the periosteum—periosteal sarcoma—or within the bone—central or endosteal sarcoma. Of the two situations the periosteal is by far the more common, but in many specimens the extent of the growth within and without the bone renders accurate determination of its site of origin impossible.

Periosteal sarcoma may arise from either the deep or superficial aspect of the periosteum. In the former case, which is the more common, it grows round the circumference of the bone between the periosteum and the outer surface of the compact layer, so that a layer of tumour substance, in which two or three faint concentric lines of new bone may be formed, occupies the space between the two. These concentric rings of new subperiosteal bone may be seen both in transverse sections through the tumour and in X-ray photographs, and serve to establish the point of origin of the growth in relation to the periosteum. Apart from this layer no distinction can be drawn between tumours which arise from the deep and superficial aspect of the periosteum. In both the sarcoma tends to spread round the bone to a greater extent than along its axis until a ring of growth is formed the edge of which rises abruptly from the surface of the shaft. This ring is only completed comparatively late in the history of the case, and for a considerable time the continuity of the shaft can be traced into and along a groove in the tumour diametrically opposite to the point of origin.

The surface of a periosteal sarcoma is lobulated, partly owing to irregularity in growth and partly because tendons and fibrous intermuscular septa offer greater resistance to destruction than do the fleshy parts of muscles, and therefore come to occupy grooves on the surface of the tumour. The surrounding muscles are destroyed and replaced, and, while they are adherent to the surface of the sarcoma because infiltrated by it, infiltration is so quickly followed by complete replacement that the line of demarcation between tumour and muscle is always well defined.

Erosion of the bone proceeds simultaneously with invasion of the soft parts, but at a slower rate commensurate with the greater density of the tissue involved. When the compact layer has been penetrated the tumour spreads along the medullary cavity and may extend as far as or beyond the external limits of the growth. The bone may be so weakened that spontaneous fracture occurs.

A section through a periosteal sarcoma in which intercellular substance is formed in sufficient quantity shows that growth occurs along radiating lines at right angles to the surface of the bone, and in cases where the intercellular substance undergoes calcification or ossification these radiating lines constitute a prominent feature in a skiagram.

Central sarcoma by its centrifugal growth erodes the inner surface of the compact bone. The stimulus provided by the presence of the tumour and by the inability of the weakened bone to withstand mechanical strain evokes a compensatory deposition of new subperiosteal bone—the phenomenon known as ‘expansion’. The intensity of the stimulus to expansion decreases gradually beyond the edge of the tumour, and the swelling therefore rises gradually from the normal surface of the shaft, in contrast with the abrupt edge of a periosteal growth. As the destructive force of the sarcoma is greater than the reparative power of the periosteum, the bone surrounding the growth becomes progressively thinned and ultimately perforated. The further development of the tumour then resembles that of a periosteal sarcoma. A central sarcoma may extend in the medullary cavity considerably beyond the limits of the expanded portion of the bone.

The term ‘parosteal’ is applied to a sarcoma which, while sufficiently close to a bone to affect its structure, does not certainly arise from the periosteum.

Sarcoma of bone, whether of central or periosteal origin, is classified according to the shape of its constituent cells into round-cell, spindle-cell, or mixed-cell sarcoma, and, according to their arrangement, as plexiform, endothelial, and peri-angial sarcoma. According to the nature and arrangement of the stroma or of the intercellular substance produced by the activity of the tumour cells, it is classified as lympho-, myxo-, fibro-, chondro-, or osteo-sarcoma.

In all cases the blood-vessels are embryonic in character, with walls formed often of a single layer of endothelium which may be indistinguishable from the cells of the tumour.

The cellular types of sarcoma, whether round-, spindle-, or mixed-celled, present no characters by which the shape of their constituent cells can be determined on naked-eye inspection, and their appearance depends largely on the degree to which degenerative changes have occurred. They are usually soft and of a white or pink colour at the growing edge, which is clearly marked off from the surrounding tissues, except in the case of the marrow, which it may resemble closely in appearance. The edge is often surrounded by an incomplete fibrous capsule which is firmly incorporated both with the surface of the tumour and with the surrounding structures.

When stroma or intercellular substance is formed in sufficient quantity, the nature of the tumour is readily appreciable to naked-eye examination of

a transverse or longitudinal section. The quantity formed of fibrous tissue, cartilage, or bone, varies from an amount which gives a definite naked-eye or radiographic character to the tumour down to one so small as to be appreciable only to microscopic investigation. In central fibro-sarcoma the arrangement of the fibrous tissue closely resembles that of a simple fibroma. In periosteal fibro-sarcoma the fibrous tissue is commonly arranged in radiating lines extending out from the surface of the bone towards the periphery of the growth. Abundance of fibrous tissue in a sarcoma is usually associated with a relatively low grade of malignancy, whereas in the ossifying variety of sarcoma no such association between structure and destructive power obtains.

In chondro-sarcoma the cartilage may take the form of scattered islands lying in an undifferentiated groundwork of cellular sarcoma, or may form the main bulk of the tumour, which is then only distinguishable from a simple chondroma by microscopic examination or by the clinical course of the case. The formation of cartilage and bone frequently proceeds side by side, the cartilage becoming calcified and undergoing transformation into bone of normal histological appearance. In other cases the intercellular substance in which the tumour cells are embedded becomes calcified without the production of regular canalicular systems. To such tissue the term 'osteoid' has been applied.

The most common form of sarcoma is the spindle-celled periosteal growth in which both cartilage and bone are formed. The bone is usually laid down in lines radiating from the periosteal surface into the tumour between the columns of cells of which the growth is composed. In quantity it is sufficient to impart the character of bony hardness and to be readily appreciable on X-ray examination or to naked-eye inspection of a transverse or longitudinal section. In the macerated specimen it is often so abundant as to form a complete skeleton of the tumour.

The secondary deposits from the more elaborately constituted varieties of sarcoma may repeat the structure of the primary growth, but have, in general, a tendency to revert to simpler cellular types.

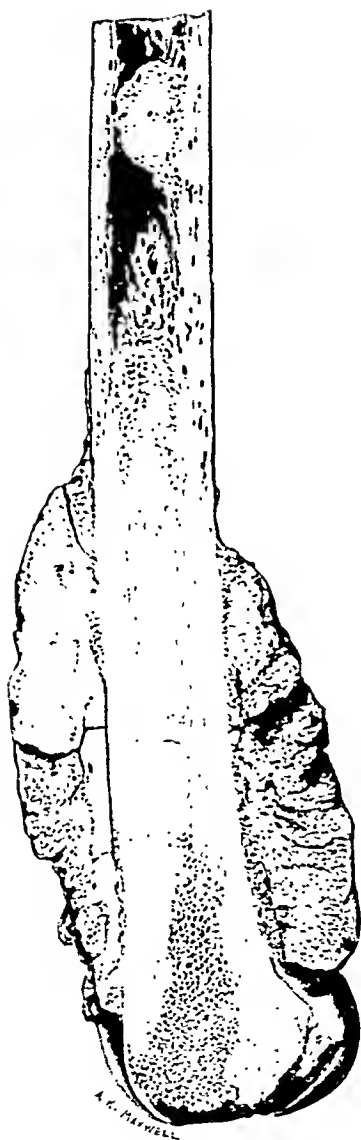
The microscopic structure of sarcoma of long bones is similar in essentials to that of sarcoma occurring elsewhere. Round-cell sarcoma is composed of small round cells of uniform size and shape, bearing a general resemblance to the small mononuclear cells of the blood, with a small quantity of homogeneous intercellular substance between them. In spindle-cell sarcoma the cells and their nuclei are elongated and arranged in irregularly branching bundles running in various directions, so that in any one microscopic field some appear in longitudinal, others in transverse, section. In both round- and spindle-cell sarcoma multi-nucleated giant cells tend to appear, scattered singly or in small groups throughout the section. The term mixed-cell sarcoma is applied to any sarcoma the size and shape of whose constituent cells is not sufficiently homogeneous to allow of inclusion among the round- or spindle-cell variety. Reference has already been made to the character of the blood-vessels and to the formation of more highly elaborated types of connective tissue in sarcomata.

The naked-eye and microscopic appearances of sarcomata on section are

often modified by hæmorrhage occurring into their substance, though the frequency with which this occurs is exaggerated in the case of museum specimens removed by amputation, owing to mechanical injury sustained during the operation. Aseptic necrosis is frequent in the older parts of tumours, and inflammation and œdema are occasional causes of variation from the original appearance of the growth.

Among the varieties of degeneration which modify the structure of sarcoma, mucinoid, fatty, and calcareous are the chief. Cyst formation is a frequent sequel of hæmorrhage or degenerative change.

Metastasis from a primary sarcoma of bone takes place mainly through the blood-stream, and the secondary growths usually appear first in the lungs and in the bones of the skull. As a rule they repeat the structure of the primary growth, but may, in the case of the more complex forms, revert to the primitive cellular type from which they were derived. Entrance to the circulation is facilitated by the free circulation of blood through the vascular spaces of the tumour, less commonly by direct growth of the sarcoma into the lumen of a large vein. In addition to metastasis by the blood-stream, secondary deposits develop from cells carried by the lymphatics to the regional lymph-glands. The same repetition of the original structure of the primary growth is seen.



### OSTEO-SARCOMA OF FEMUR.

A sagittal section of the lower half of a femur, macerated.

The lower part of the shaft is surrounded by a fusiform mass of bone, which represents the skeleton of a sarcoma. The medullary cavity is filled by dense bone opposite to and slightly above the external growth. Near the upper end of the specimen a hemispherical mass of bone in the medullary canal marks the site of a detached portion of the tumour.

### PERIOSTEAL OSTEO-CHONDRO-SARCOMA OF ULNA.

A vertical section of the upper half of an ulna with portions of the radius and lower end of the humerus removed by amputation.

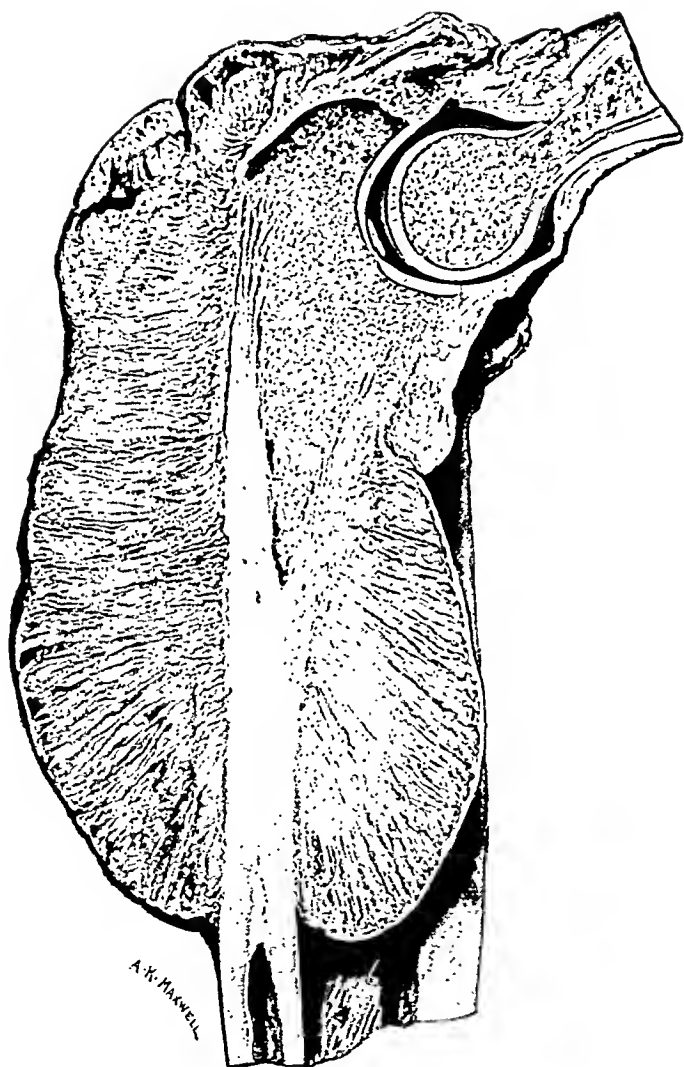
The upper end of the ulna is surrounded by a periosteal sarcoma which has invaded the cancellous tissue. The cut surface of the tumour is marked by parallel lines of calcified substance arranged, for the most part, at right angles to the bone.

*Hunterian Museum, R.C.S. 1661.1*

MICROSCOPIC STRUCTURE.—The tumour is a round-celled sarcoma, in which small islands of cartilage and more extensive areas of osteoid tissue occur; here and there the calcified material has the appearance of true bone.

CLINICAL HISTORY.—The patient was a man, aged 24, in whom the tumour had grown slowly for two years. He recovered after amputation.





## PERIOSTEAL. OSTEOID-CHONDRO-SARCOMA OF TIBIA.

A sagittal section of the upper two-thirds of a right leg.

Projecting from the front of the tibia, immediately below the insertion of the ligamentum patellæ, there is a hemispherical subperiosteal tumour 7.5 cm. in vertical diameter. The bone behind is only superficially involved. The tumour contains a conspicuous amount of cartilage, as well as fine pale yellow lines indicative of decalcification. *Hunterian Museum, R.C.S. 1656.1*



× 61

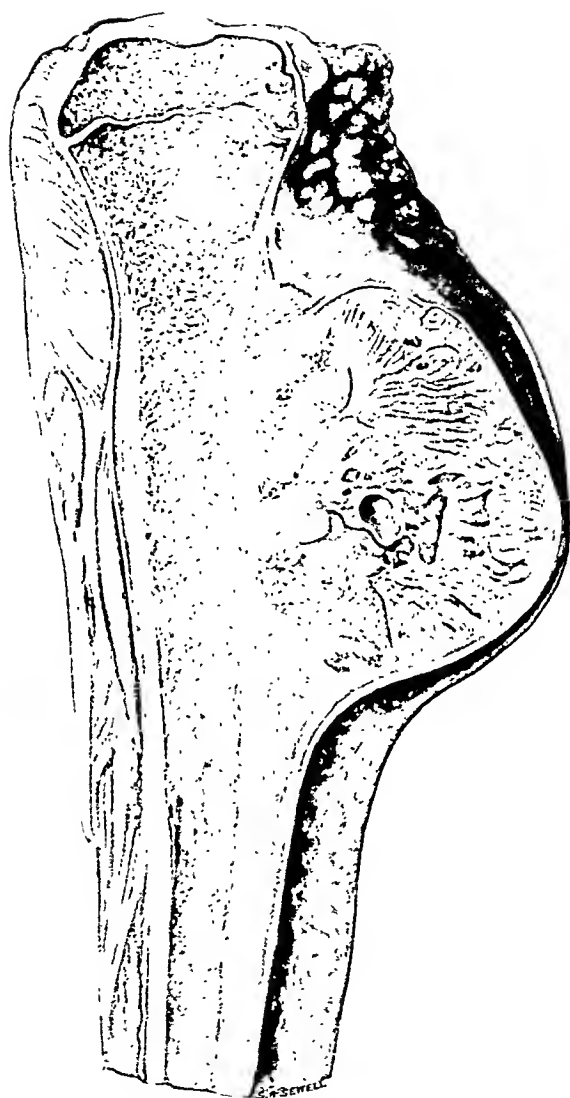


× 267

**MICROSCOPIC STRUCTURE.**—The tumour is a chondro-sarcoma in which osteoid tissue is in process of formation, i.e., tissue produced by the calcification of the intercellular substance in which the tumour cells are embedded, without formation of canalicular systems.

**CLINICAL HISTORY.**— The limb was removed from a girl, aged 13, who had received a blow on the right leg nine months previously. Swelling occurred a month later and increased rapidly. The tumour was incised three months before amputation on the supposition that it was a tuberculous abscess.

1914.



HUNTERIAN MUSEUM, R.C.S. 1656.1

## CHONDRO-SARCOMA OF FEMUR, ASSOCIATED WITH OSTEITIS DEFORMANS.

A sagittal section of the lower half of a right femur.

A chondro-sarcomatous tumour has grown round the lower end of the femur, which is enlarged and bent forwards in consequence of osteitis deformans. The bone is extensively destroyed by the new growth, which has invaded the medulla and is running for some distance up the centre of the shaft. The greater part of the articular cartilage is not affected.

*Hunterian Museum, R.C.S. 1879.1*

**CLINICAL HISTORY.**—The patient was a kyphotic woman, aged 67, who, in December, 1910, was admitted to hospital, where her thigh was amputated. She had felt shooting pains in the left leg for ten years, and found that the skin was thickened. Six months later pain was felt in the right leg. Both legs and thighs subsequently became bent and she thought her head was growing larger. The pain in head and limbs was continuous and worse at night.

A painful swelling, which increased in size, appeared round the right knee five months before admission to hospital.

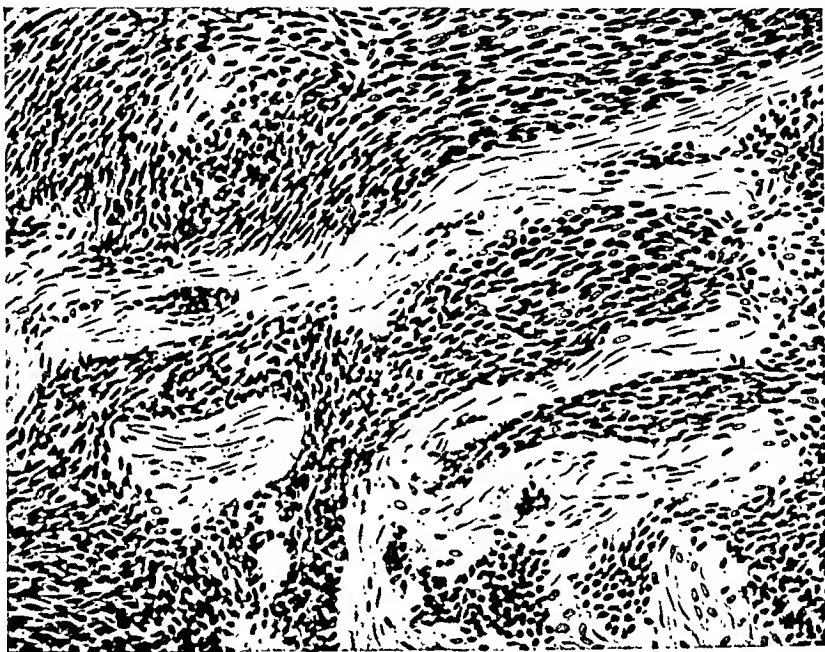


HUNTERIAN MUSEUM, R.C.S. 1379.1

## PAROSTEAL PLEXIFORM SARCOMA OF TIBIA.

A longitudinal section of the upper half of a tibia, with the soft parts on the front and inner aspect.

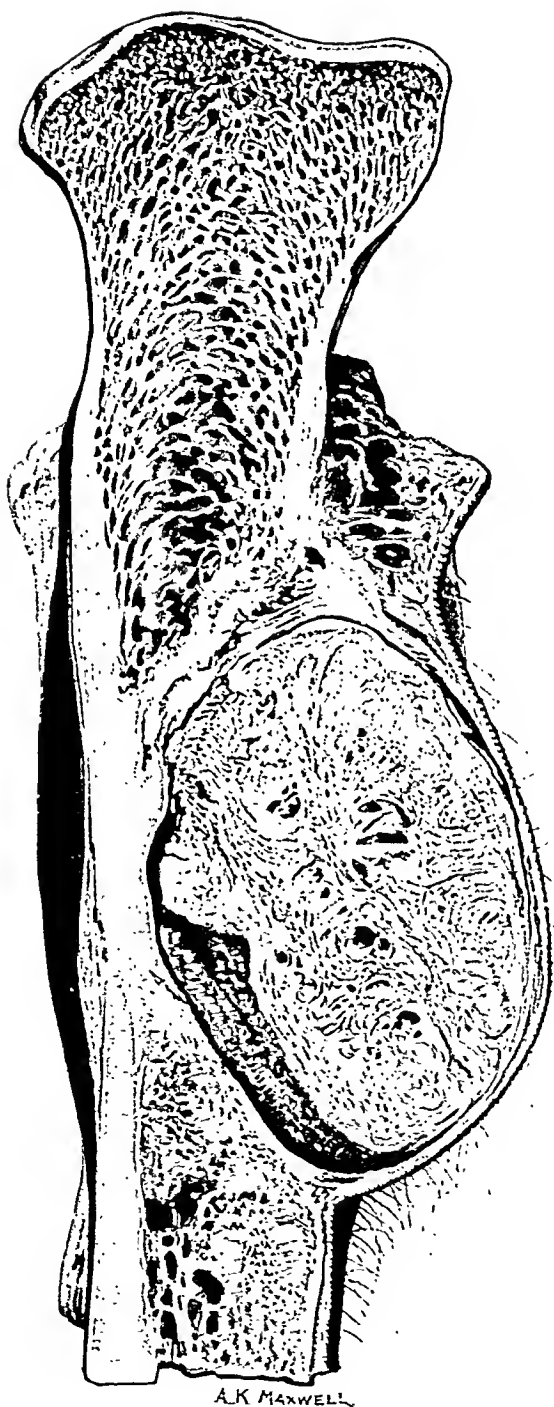
Lying over the anterior surface of the shaft of the bone and half embedded in it, is an oval tumour 7·5 em. in its chief vertical diameter. The suprajacent skin is not invaded, nor is the osseous tissue beneath the growth. The irregularity on the posterior aspect of the bone is due to a fracture which followed a local operation. *Hunterian Museum, R.C.S. 1677.1*



× 184

**MICROSCOPIC STRUCTURE.**—The neoplasm consists of a plexus of branching cell columns separated by well-defined septa of fibrous tissue. The groups of cells vary much in size; some are quite narrow and of considerable length; others are more voluminous and their component cells, which are spindle-shaped, are disposed in intersecting bundles as in a spindle-celled sarcoma. The growth may be regarded as a plexiform sarcoma.

**CLINICAL HISTORY.**—From a man who, in 1865, when a boy, was severely kicked on the shin of the right leg. At or near the spot there formed a lump which slowly grew, and hurt after much walking. In 1877 the part was again severely struck and became more tender. The mass then seemed to lie between the skin and the bone; but in the beginning of 1880 a swelling like hard bone appeared beneath it. In June, 1883, a tumour, which was embedded in the bone, was excised. In October the patient fell and broke the leg near the seat of the operation. Soon afterwards recurrence occurred in the lower part of the scar, and the growth was removed for the second time in February, 1884. It recurred in January, 1885, and the following year amputation was performed.



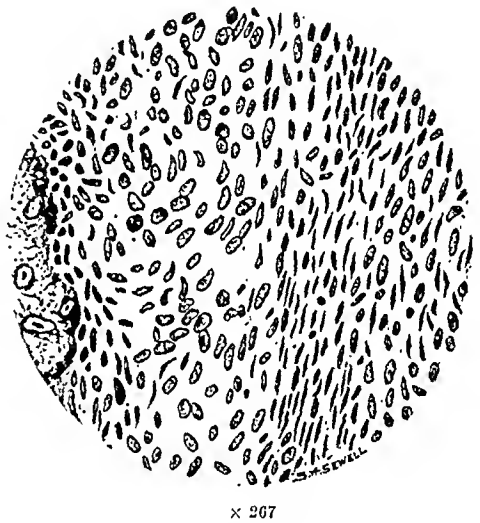
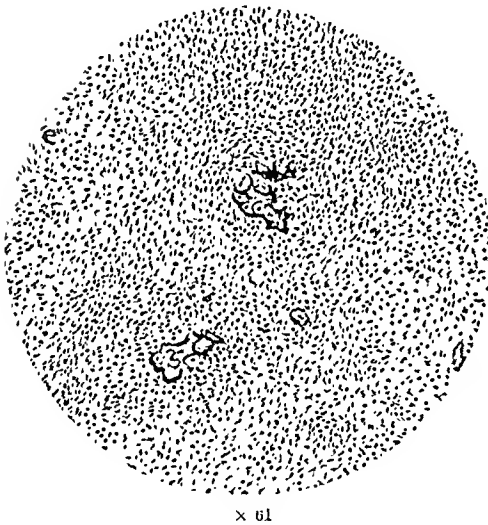
A.K. MAXWELL

## OSTEOID-CHONDRO-SARCOMA OF FEMUR.

A coronal section of the lower half of a femur.

The shaft and lower extremity are surrounded and invaded for a distance of 14 cm. by a dense, white sarcoma. The periosteal portion of the tumour has a nodular surface and is traversed by white lines of calcified material which project outwards at right angles to the bone. Immediately above the inner condyle it consists largely of hyaline cartilage. The endosteal part of the tumour has replaced completely the cancellous tissue and medulla of a corresponding part of the bone. The compact cortex of the shaft has been eroded on its medullary aspect to half the normal thickness.

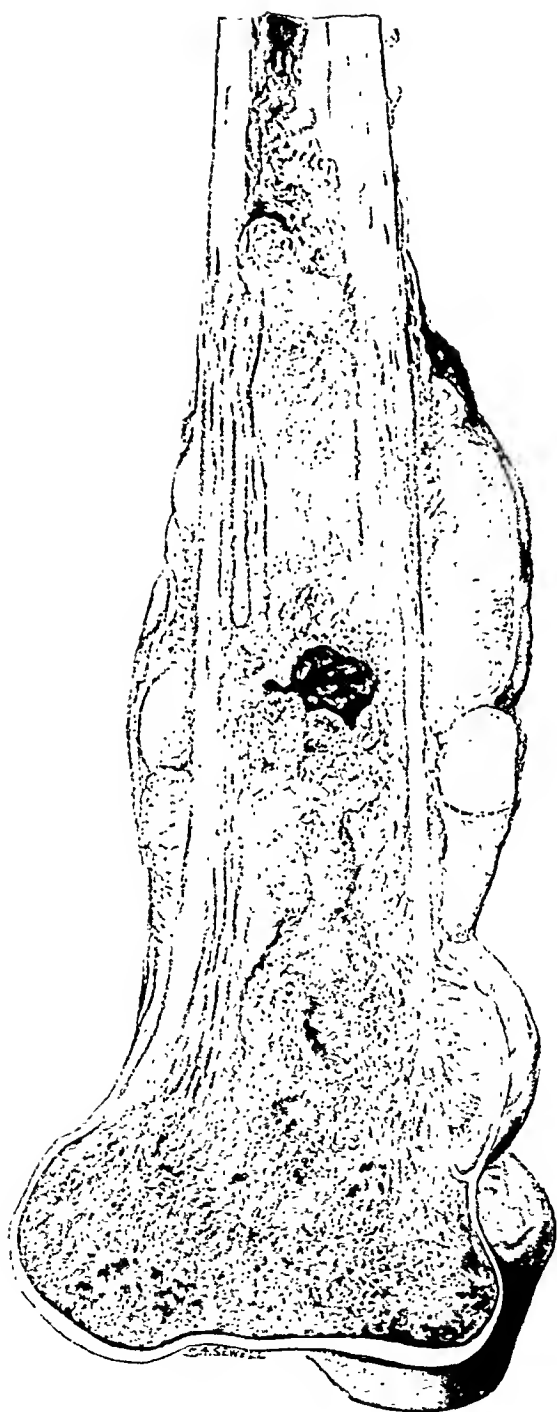
*Hunterian Museum, R.C.S. 1654.1*



**MICROSCOPIC STRUCTURE.**—Spindle-celled sarcoma with cartilage formation. The cartilage is extensively calcified.

**CLINICAL HISTORY.**—The patient was a woman, aged 25, who had complained of dull pain about the right knee-joint for three months, and had been treated for synovitis. There was no history of injury. The knee-joint contained a small quantity of fluid. Its movements were normal. There was no evidence of disease in the inguinal glands, abdomen, or thorax. In March, 1909, after a piece of growth had been removed for examination and found to be sarcomatous, the limb was amputated at the hip-joint. The patient remained free from local recurrence, but signs of secondary growth appeared in the lungs in February, 1910. A post-mortem examination was not obtained.





HUNTERIAN MUSEUM, R.C.S. 1654.1

## CENTRAL ROUND-CELLED SARCOMA OF FEMUR.

A sagittal section of the lower end of a femur enlarged for a distance of 11.5 cm. by the growth of a central tumour, the irregular patches of opacity in which are due to necrosis.

The upper limit of the pale semi-translucent growth is well defined ; it lies in immediate contact with the cancellous tissue, and is devoid of any kind of capsule. The articular cartilage is intact, although over the trochlea the neoplasm is almost in contact with its deep surface.

*Hunterian Museum, R.C.S. 1570.2*

MICROSCOPIC STRUCTURE.—Round-celled sarcoma.

CLINICAL HISTORY.—From a woman who fell on her knee three months before and had lost two stone in weight since the accident. On examination there was effusion into the joint, with a diffuse swelling of the lower end of the femur ; the lymphatic glands were not involved. Amputation was performed in 1919. In October, 1924, metastases were present in the lower jaw and frontal bone.

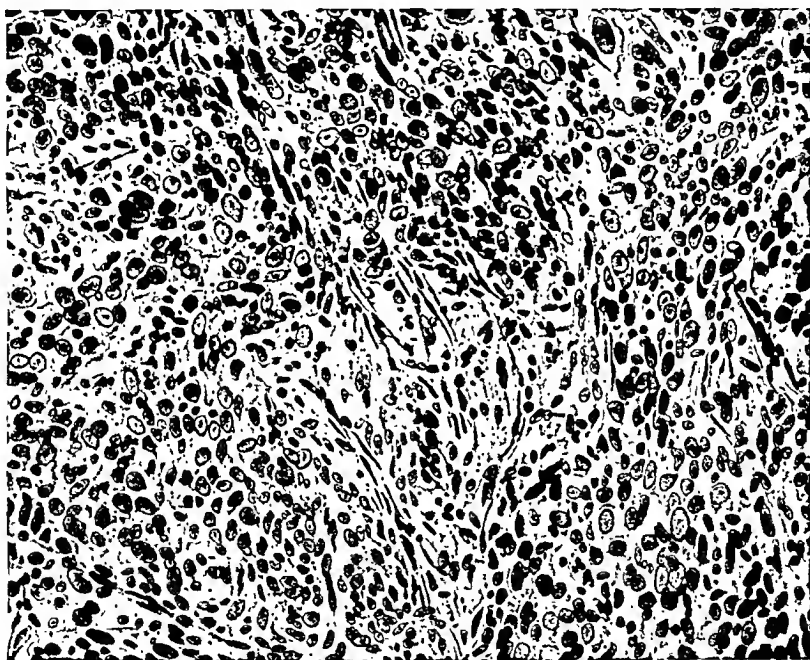


## CENTRAL MIXED-CELLED SARCOMA (ROUND, SPINDLE, AND GIANT) OF FEMUR.

A coronal section of a knee-joint.

The lower end of the femur has been destroyed by a large spheroidal tumour, into which hæmorrhage has taken place. The swelling projects chiefly from the inner aspect. Both condyles are in large part destroyed, but retain most of their articular cartilage intact. Between them the growth has commenced to involve the crucial ligaments. To the naked eye the substance of the tumour is almost structureless owing to degeneration.

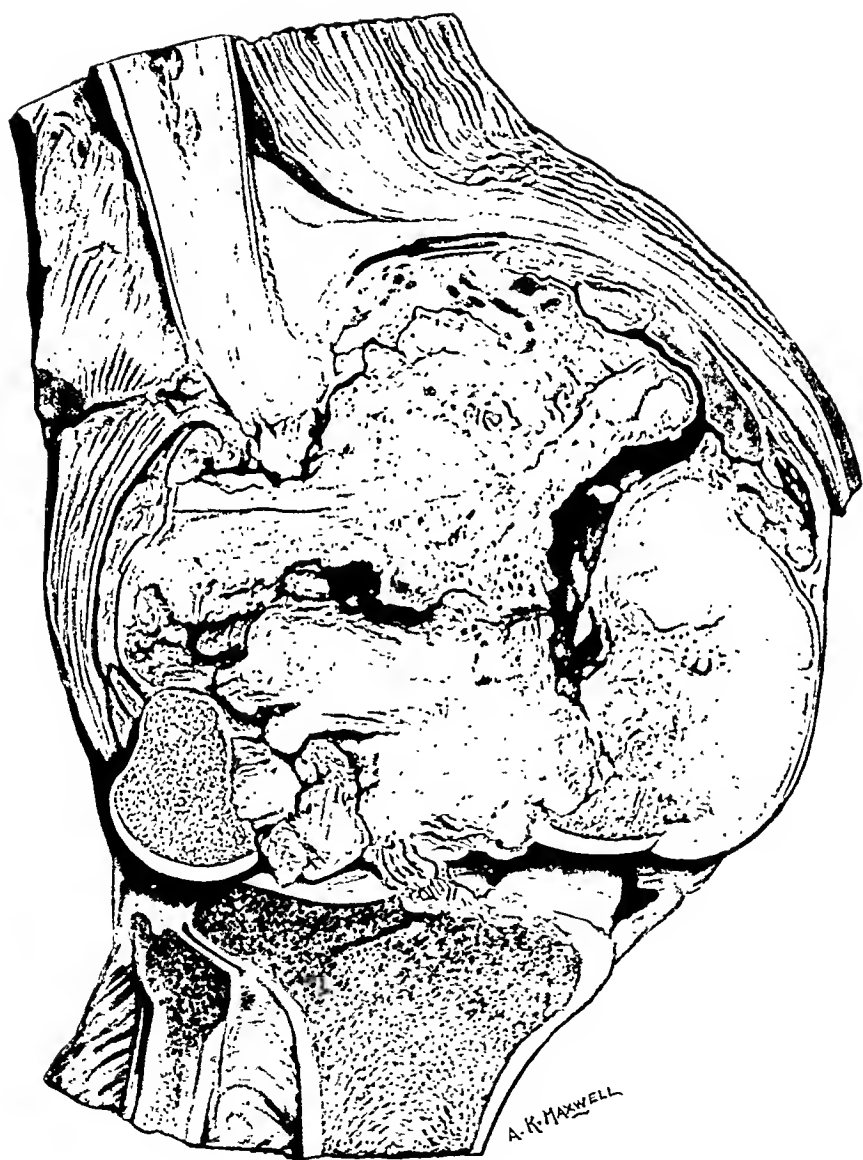
*Hunterian Museum, R.C.S. 1618.1*



x 181

**MICROSCOPIC STRUCTURE.**—The more solid portions of the growth projecting from the inner side consist of pure spindle-celled sarcomatous tissue; in other parts the spindle cells are a little elongated, and there occur areas in which the cells are round; multinucleated giant-cells are scattered through the growth.

**CLINICAL HISTORY.**—The parts were removed by amputation from a man, aged 23, who had received an injury to the right knee four months previously; he was off work one day only. Two months later a painful enlargement was observed on the inner side of the joint; this increased rapidly, and was especially painful at night. X-ray examination showed displacement backwards of the tibia. The femoral glands were not involved.



## CENTRAL FIBRO-SARCOMA OF FEMUR.

A vertical section of the lower end of a femur. For a distance of 11 cm. the bone has been completely destroyed by the growth of a central tumour composed largely of dense fibrous tissue. The articular cartilage remains intact.

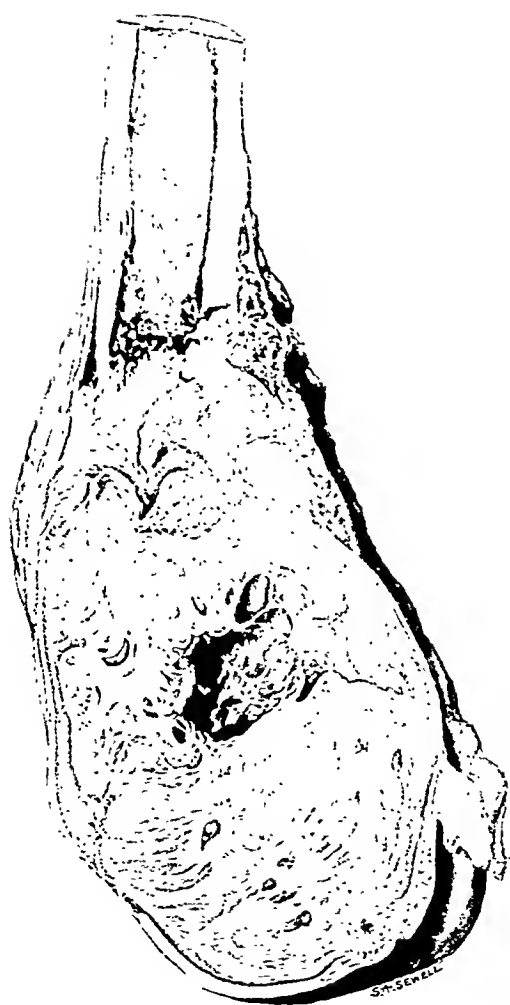
Posteriorly the growth is not only circumscribed but invested with a stout cohering capsule, probably derived from the periosteum; superiorly and anteriorly this is wanting, the neoplasm lying in direct contact with the medulla of the shaft and with the fat over the front of the swelling. To the naked eye the tumour consists of interlacing bands of white fibrous tissue, which in the lower part are undergoing mucinoid degeneration, and centrally have been completely destroyed, with the resulting formation of an ill-defined cavity filled with mucus. *Hunterian Museum, R.C.S. 1629.2*



### MICROSCOPIC STRUCTURE.—Fibro-sarcoma.

**CLINICAL HISTORY.**—From a patient, aged 23, who complained of occasional pain for one year and swelling of the lower end of the femur for about six months. When seen in September, 1919, there was a firm fusiform swelling on the antero-external aspect of the lower end of the right femur. Portions of the periphery and centre of the tumour were removed for microscopical examination, and were thought to consist of dense fibrous tissue, with no evidence of malignancy.

On Sept. 25, 1919, the tumour was enucleated through an incision on the inner side of the thigh. It appeared to be completely surrounded by a dense, fibrous capsule, and extended upwards into the shaft of the femur, and downwards into the cancellous tissue of the condyles. Although tough, the tumour was enucleated without much difficulty, leaving a shell of the

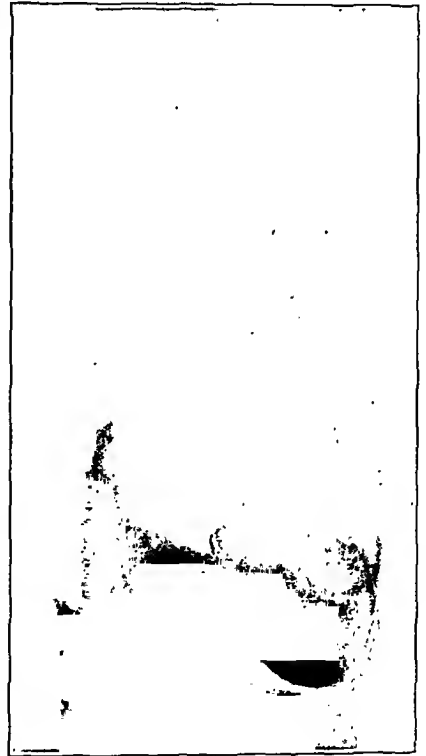


HUNTERIAN MUSEUM, R.C.S. 1629.2

bone anteriorly, the whole of the posterior part of the lower third of the shaft being destroyed. Recovery was uneventful, and the patient was soon able to walk with a caliper splint.

Skiagrams taken in May, 1920, showed no evidence of recurrence. Others taken in September, 1920, however, disclosed an upward spread of the disease, although there was no external evidence of it. In March, 1921, there were definite clinical and radiographic signs of recurrence; the lower end of the femur was thickened, the knee slightly flexed, and the joint contained fluid.

Amputation was carried out through the middle of the thigh, March 21, 1921.





CENTRAL CHONDRO-SARCOMA OF HUMERUS.



*By permission of Dr. W. H. Coldwell.*

HUNTERIAN MUSEUM, R.C.S. 1639.5

NO 2—SUPPLEMENT

B2

## CENTRAL CHONDRO-SARCOMA OF HUMERUS, *continued.*

A vertical section of the upper half of a left humerus.

The greater part of the upper extremity of the bone and the adjoining portion of the medullary cavity of the shaft are occupied by a white growth which has expanded the head and neck. Its surface is marked by many small cysts which have resulted from local softening. At the surgical neck is a fracture which was produced by the operation.

*Hunterian Museum, R.C.S. 1639.5*



**MICROSCOPIC STRUCTURE.**—Chondrifying spindle-cell sarcoma.

**CLINICAL HISTORY.**—The patient was a man, aged 48, whose left shoulder first became painful in January, 1919. By April the pain had so increased in severity as to prevent sleep and had extended down the arm. At that time the deltoid was wasted, the head of the humerus was enlarged, and movement at the shoulder-joint was both painful and limited. The limb was removed by interseapulo-thoracic amputation in June, 1919. He remained free from symptoms until October, 1921, when he spat blood for four days. After this he was quite well until June, 1923, when a lump appeared on the right side of the forehead. It was explored and found to be a secondary growth eroding the frontal bone. This tumour was still the only recognized metastasis in October, 1924, and, apart from headache, the patient had retained good health.



HUNTERIAN MUSEUM, R.C.S. 1639.5

## OSTEOID-CHONDRO-SARCOMA OF TIBIA

(METASTASIS IN SYNOVIAL MEMBRANE OF KNEE-JOINT AND IN FEMUR).

A vertical section of a left knee-joint. The upper end of the tibia is largely replaced and partially surrounded by a growth which is clearly differentiated from the surrounding tissues. The bone is not expanded and the articular cartilage is intact, though the tumour is in contact with its deep surface. The compact layer of the posterior surface of the tibia has been eroded by the growth a short distance below the joint, so that the parts within and without the bone are here continuous. The popliteus muscle has been replaced by the backward extension of the tumour, which presents a lobulated surface towards the popliteal vessels.

The sarcoma is of an opaque, yellowish-white colour with a central mass of hyaline cartilage, and presents various stages of calcification.

There is a similar but much smaller growth in the cancellous tissue of the lower end of the femur. From the synovial membrane immediately below the patella projects a plano-convex pendulous body which has the same structure, both macroscopic and microscopic, as the tumours referred to, and represents a local metastasis in one of the synovial fringes. A second pendulous body with precisely similar characters projects from the back of the joint against the condyle of the femur. *Hunterian Museum, R.C.S. 2062.1*

**MICROSCOPIC STRUCTURE.**—The central portion has the typical appearance of hyaline cartilage, the cells being spherical, of large size, and widely separated by tracts of hyaline matrix in which no calcification has occurred. For some distance around this the tissue within the bone is densely and uniformly calcified. The subperiosteal extension shows areas of true cartilage which merge into and mingle with areas of sarcomatous tissue.

**CLINICAL HISTORY.**—The limb was amputated from a lady, aged 74. In the spring of 1899 she noticed that the left knee was gradually swelling. The enlargement was accompanied by pain, especially on movement and at night, and osteo-arthritis had been diagnosed in the first instance. The swelling continued to increase, and in October, 1899, was recognized as sarcomatous. The operation was successful.

(*S. G. Shattock, Trans. Pathol. Soc., 1901, ii, 275.*)



A. K. MAXWELL

H. STERAN MUSEUM, R.C.S. 2062.1

NO. 2—SUPPLEMENT

B1

## SUBPERIOSTEAL LYMPHO-SARCOMA OF TIBIA.

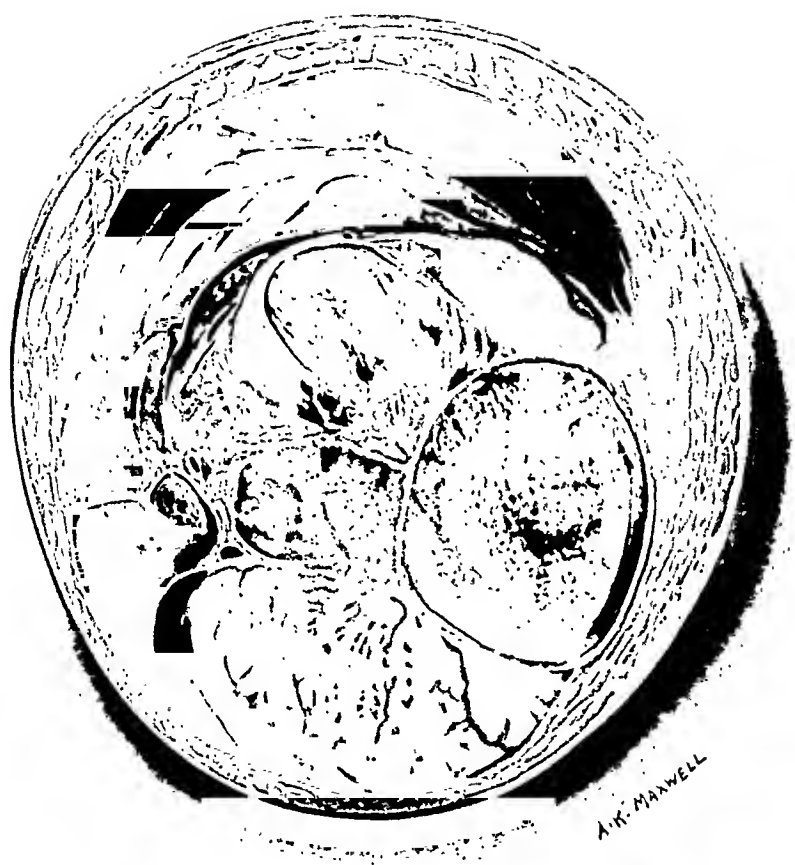
A transverse section of the right leg.

The tibia is enlarged by a tumour which has grown round the whole circumference between the periosteum and the surface of the shaft, and in this situation presents faint concentric rings of newly-formed bone. The growth has eroded the periphery of the shaft but has not invaded the medullary cavity. From the anterior, lateral, and posterior aspects of the bone the tumour projects into the surrounding muscles. It is of an opaque white colour, lobulated on the surface, and clearly differentiated from the normal muscle which remains.

*Hunterian Museum, R.C.S. 1581.1*

MICROSCOPIC STRUCTURE.—Lympho-sarcoma.

CLINICAL HISTORY.—The patient was a boy, aged 11 years, in whom a painful and tender swelling of the right leg had been growing for one year. On examination, the upper third of the shaft of the tibia was thickened over its inner surface. From its outer aspect an elastic, lobulated tumour projected into the soft parts of the leg. The superficial veins were dilated. The inguinal glands were not enlarged. Supracondylar amputation was performed in November, 1919.



HUNTERIAN MUSEUM, R.C.S. 1581.1

SUBPERIOSTEAL LYMPHO-SARCOMA OF TIBIA, *continued.*



A.K. MAXWELL.

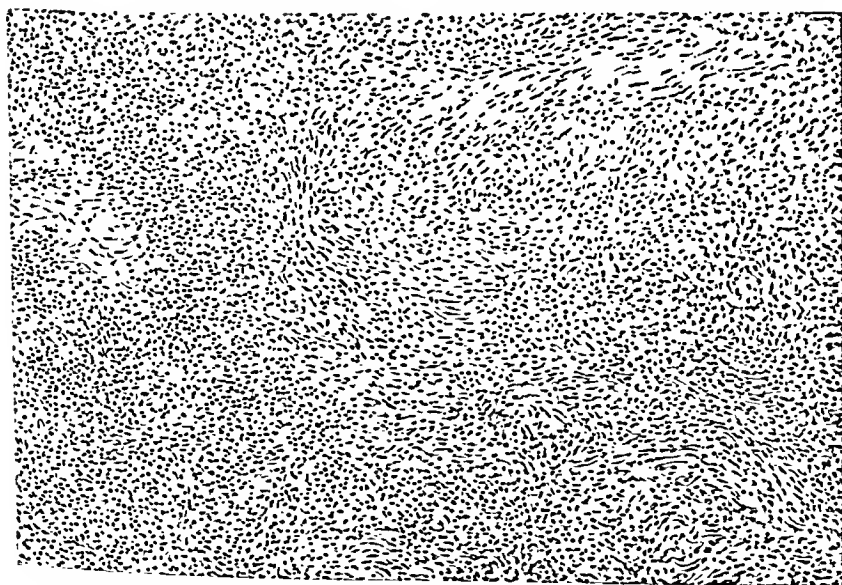
x 97



# CHONDRO-MYXO-SARCOMA OF FEMUR.



(1)  
x 80



(3)  
x 80

### CHONDRO-MYXO-SARCOMA OF FEMUR, *continued.*

A sagittal section of the lower end of the right femur, with the patella and some of the soft parts.

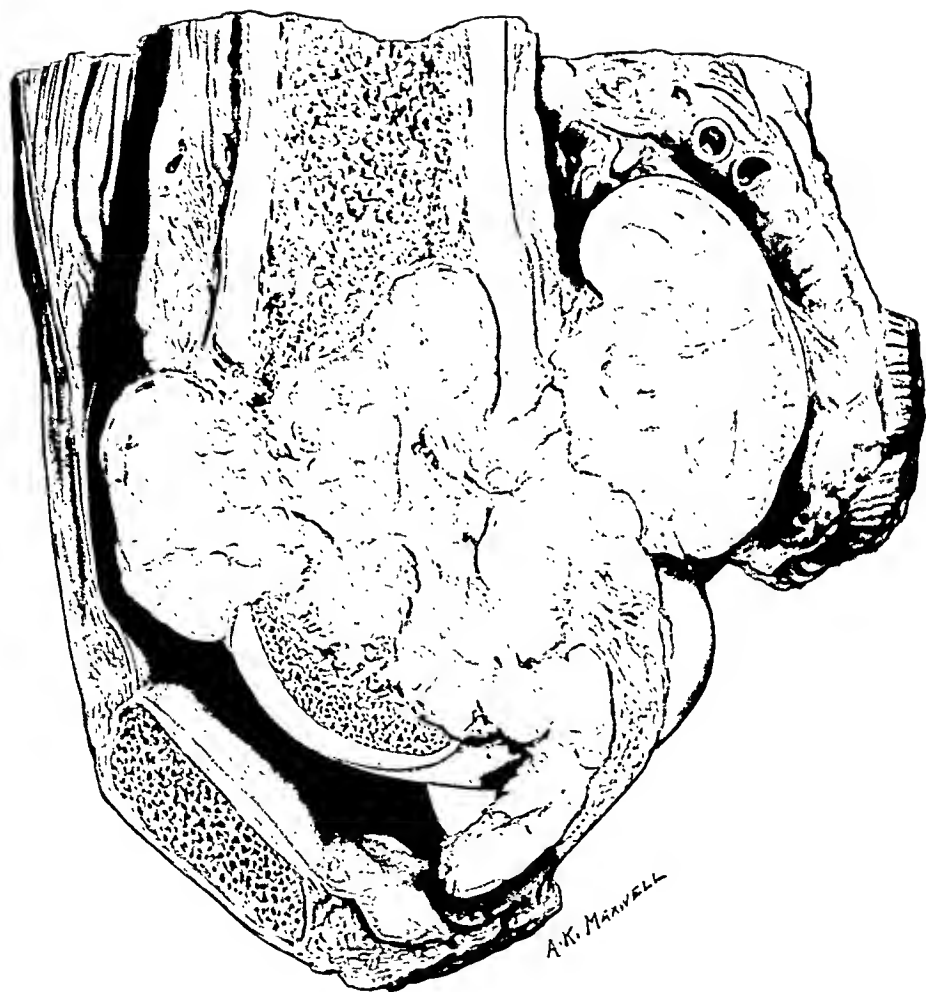
The cancellous bone of the lower end of the femur has been replaced by a lobulated tumour which consists largely, though not entirely, of hyaline cartilage. The growth has perforated the compact bone at three points, from which it projects forwards into the knee-joint, backwards into the popliteal space, and downwards along the crucial ligaments.

*Hunterian Museum, R.C.S. 1639.1*

**MICROSCOPIC STRUCTURE.**—Some parts of the tumour have the appearance of a simple myxo-chondroma. In other parts the cells are abundant, with little or no intercellular substance, and the tumour has the characters of a sarcoma. (*See* p. 33.)

**CLINICAL HISTORY.**—The patient was a woman, aged 30, whose right thigh was amputated in 1894 for a swelling at the knee of three months' duration. Before operation the tumour was regarded as a cystic giant-celled sarcoma. For the previous ten months she had been treated by massage for general weakness of the right lower limb, but no lump was noticed during this time.

The patient remained well for four years, and then died of a sarcoma of the brain.



HUNTERIAN MUSEUM R.C.S. 1639.1

## II. SIMPLE TUMOURS OF BONE.

THE varieties of simple tumour specially connected with bone are chondroma, osteoma, and myeloma, representing respectively cartilage, bone, and marrow. Fibroma occurs almost exclusively in the jaws. In addition to these, any kind of simple tumour of connective-tissue origin may occasionally grow in relation to a bone.

**CHONDROMA.**—A chondroma is an encapsuled tumour composed of hyaline cartilage. Where a definite origin can be traced, this is always from epiphysial—never from articular—cartilage. The tumour may arise from any bone, and tends to grow outwards from the surface rather than within its substance. The majority of cartilaginous tumours which spring from long bones ossify as they grow, and this variety will be described under the heading **OSTEOMA** (p. 37).

Chondromata which remain wholly or mainly cartilaginous throughout their life-history may occur as single or multiple tumours, but, while the single variety may be found in connection with several parts of the skeleton, the multiple type affects almost exclusively the long bones of the hand and the foot.

The single chondroma of long bones commences in the neighbourhood of an epiphysial cartilage, to which, or to a sequestered fragment of which, its origin is commonly attributed. It grows both within and without the bone to which it is attached, but chiefly tends to spread outwards from the periosteal surface, and only in rare cases causes appreciable expansion of the shaft. In time it may completely encircle the bone, and, since it is active in growth and productive of but trivial symptoms, it may form a swelling of striking size in individuals to whom the mere presence of an unexplained lump is not an incentive to seek treatment.

The surface of the tumour is notably lobulated, its consistence is elastic, and its appearance on section is that of lobules of translucent cartilage bound together by a framework of fibrous tissue. This appearance is frequently modified by the presence of opaque white areas of calcification and by cysts caused through myxomatous changes in the cartilage.

A single chondroma of similar character may also grow from the bony wall of the thorax or pelvis. When growing from a rib its projection into the cavity of the chest or abdomen is usually much greater than the surface prominence would suggest.

Chondromata are not uncommon in the metacarpals and in the phalanges of the fingers, and occasionally occur in the corresponding bones of the foot. They are usually multiple, and, when multiple, are always central in position, so that they expand the bones in which they grow. As in the case of the larger long bones, they arise from the region of the epiphysial cartilage, and are therefore found near the heads of the metacarpals and the bases of the phalanges. Great distortion of the skeleton of the hand may result from

their growth. Calcification is the only structural change which occurs in these tumours.

From a consideration of clinical histories it would appear probable that the single chondroma of long bones has a slight, but definite, tendency to sarcomatous change in the course of years. In this connection it may be recalled that the distinction between a simple chondroma and a sarcoma which produces cartilage in bulk can only be established by precise microscopic examination of the tissue which separates the cartilaginous masses.

**OSTEOMA.**—An osteoma is a non-malignant tumour composed of bone. Both cancellous and compact varieties occur.

*Cancellous Osteoma.*—This tumour is also known as a spongy or pedunculated exostosis in reference to its structure and shape, or as an ossifying chondroma in respect of its mode of origin. It affects chiefly the long bones of the limbs, and arises during youth in the immediate neighbourhood of an epiphysal cartilage. As growth proceeds, the attachment of an osteoma is displaced progressively towards the centre of the shaft, its distance from the epiphysal line representing the amount of new bone added to the diaphysis since the first appearance of the tumour. The most common sites are those at which the most active growth occurs, and the cancellous osteoma is therefore most often found in the region of the knee, shoulder, or wrist. The habit of growth of a spongy exostosis is in agreement with its origin from a sequestered fragment of epiphysal cartilage in that increase in size of the tumour ceases with, or in many cases before, junction of the epiphysis with the shaft.

A cancellous osteoma is a pedunculated tumour with a bulbous extremity which may, in some cases, retain the nodular surface characteristic of chondromata in general. The stalk slopes gradually down to the surface of the bone on the side of the epiphysis and rises abruptly from it on the side facing the shaft. On section the tumour is composed of cancellous tissue, continuous with that of the bone from which it arises, surrounded by a compact layer and covered with a fibrous capsule continuous with the periosteum. There is usually an adventitious bursa between the tumour and the surrounding soft parts.

During the period of active growth an ossifying chondroma has a cap of hyaline cartilage, which represents the epiphysal line from which it is derived. In some cases a zone of calcification can be recognized with the naked eye in the deeper layers of this cartilage, representing the intermediate stage in the transformation of cartilage into bone.

Cancellous osteomata when growing in connection with the epiphysal scales of the scapula or iliac crest may reach a considerable size without producing symptoms. The subungual exostosis, which grows from the dorsal aspect of the terminal phalanx of the great toe and displaces the nail, is an instance of a small exostosis which may cause considerable disability. It is almost the only variety which is liable to ulcerate through the overlying soft parts.

Exostoses are often multiple, but seldom occur in large numbers unless an hereditary element is present. In this case widespread irregularity of growth in the epiphysal cartilages may produce deformity of the extremities.

*Compact Osteoma.*—The compact osteoma, or ivory exostosis, is composed

of dense bone, and arises as a sessile tumour in situations remote from cartilage. It is most commonly found on the membrane bones of the cranial vault or on the walls of cavities connected with them, such as the orbit or frontal sinus. It is a slowly-growing tumour, but may in time reach a considerable size and project within the cranial cavity as well as from the external surface of the skull.

**FIBROMA.**—Fibrous tumours of bone occur in the jaw and in the nasopharynx. In the jaw they may arise from the surface or within the substance of the bone. The common fibroma of the jaw—fibrous epulis—is a simple tumour of firm consistence and pink colour which grows from the gum in the neighbourhood of a tooth and seldom reaches any considerable size. It arises either from the periosteum of the alveolus or from the periodontal membrane, and in the latter case may remain attached to the tooth on extraction. Less commonly a fibroma is situated in the centre of the jaw and, by its growth, causes expansion of the bone. Central fibroma of the jaw shows no tendency to limitation of size.

Fibroma of the nasopharynx is a rare tumour, affecting chiefly young adult males. It arises in the periosteum of the basi-cranial axis immediately behind the choanae, and projects forwards into the nose and downwards into the nasopharynx. The tumour is of very active growth and has a strong tendency to local recurrence after removal, but does not form metastases.

**MYELOMA.**—Myeloma is a simple tumour which in the type and arrangement of its component cells bears a general resemblance to red marrow.

In conformity with its structure myeloma occurs almost exclusively in cancellous bone, either in the extremities of long bones or in the jaws. In the limbs myeloma is most common at the sites of active growth—that is, in the tibia and femur at the knee, in the humerus at the shoulder, and in the radius at the wrist—its favourite situation being the upper extremity of the tibia.

The tumour first appears in the interior of the articular extremity, though not necessarily in its exact centre, and produces expansion of the bone. Articular cartilage offers an almost absolute barrier to its passage, so that, however great the distortion of the extremity of the bone, the joint is never invaded by the tumour.

The rate of destruction of bone by a myeloma is always greater than the rate at which compensatory new bone is laid down by the overlying periosteum. Hence expansion is accompanied by gradual thinning of the bony shell until a point is reached at which the tumour comes to the surface and appears as a soft spot beyond the level of the otherwise bony-hard swelling. During the stage immediately preceding perforation, the enveloping bone may be so thin as to give the physical sign of egg-shell crackling. It is not usual for a myeloma to form a swelling of any considerable size beyond the limits of the bone in which it originates, but should this occur, the tumour displaces the soft parts by which it is surrounded. It has no power of forming secondary growths in distant parts. A myeloma of a long bone is so vascular as commonly to pulsate when it pierces its bony shell and occasionally to cause embarrassment during removal.

On section, the tumour is of a dark-red colour, soft, and rather friable.

The cut surface is seldom homogeneous throughout, but is altered by the hæmorrhages which regularly occur into its substance. Occasional white patches of tumour may be seen, and very rarely the whole myeloma may be white. The effused blood may permeate the intercellular spaces of the growth and be recognizable to the naked eye only by the colour which it imparts, or it may displace portions of the tumour and remain in a fluid state to form cysts surrounded and separated from one another by walls of fibrous or unaltered tumour substance. Bleeding may even be so constant a factor in the evolution of a myeloma that the tumour itself is represented only by a thin layer lining a cavity in the bone which otherwise is filled with fluid blood—the 'blood-cyst' of bone. Cysts may also be formed by degeneration in the substance of the tumour.

The junction of a myeloma with the bone in which it grows is well-defined and is often marked by an imperfect fibrous capsule through which project small nodules of the tumour. The latter indent the surrounding bone and produce a characteristic pitted appearance seen when the cavity is viewed from within.

The microscopic structure of myeloma is constant throughout all parts of the tumour. The characteristic feature is the uniform distribution throughout every field of large multinucleated cells of regular, round, or oval shape. Each contains from 10 to 20 nuclei, arranged irregularly, or massed towards the centre. The stroma in which the giant cells are embedded consists of small oval- or spindle-shaped cells, among which is a variable quantity of fibrous tissue. The blood-vessels are numerous and thin-walled.

Myeloma of the jaws is more common in the mandible than in the maxilla. It commences in the cancellous tissue at the base of the alveolus and extends up towards the mouth, where it forms a soft, red tumour beneath the gum. As the growth increases in size the overlying mucous membrane becomes ulcerated. While the myeloma is extending towards the mouth it is also penetrating the body of the jaw, which becomes expanded by its growth. In its most characteristic form, when fully developed, a myeloma of the mandible is of dumb-bell shape, the expanded portion of jaw being joined to the ulcerated tumour in the mouth by a narrow neck which occupies the alveolar socket of a tooth.

On section, there is usually a fibrous centre from which fibrous strands radiate irregularly towards the periphery. Between them is the soft, plum-coloured substance of the tumour. The microscopic appearance is the same as that of similar tumours of the long bones.

Myeloma occasionally occurs as a small, lobulated, encapsuled tumour, pink in colour, growing from a tendon sheath, most commonly that of a finger. Its structure is similar to that of myeloma of bone.

### CHONDROMA OF TOE.

A vertical section of the head of a first metatarsal bone with the toe.

A chondroma has grown from the terminal phalanx and projects forwards and upwards, replacing the whole of the toe with the exception of the base of the proximal phalanx. The tumour is lobulated and is traversed by fine strands of vascular connective tissue. Points of calcification are scattered over the cut surface.

*Hunterian Museum, R.C.S. 1341.1*

MICROSCOPIC STRUCTURE.—The tumour consists of hyaline cartilage.

CLINICAL HISTORY.—The patient was a man, aged 53, upon whose great toe a brick had fallen from a truck with such force as to cut the boot. The toe was amputated three years afterwards. Two months before the operation a sequestrum came away from the under side.





## CHONDROMA OF HUMERUS.

The inner half of a left humerus.

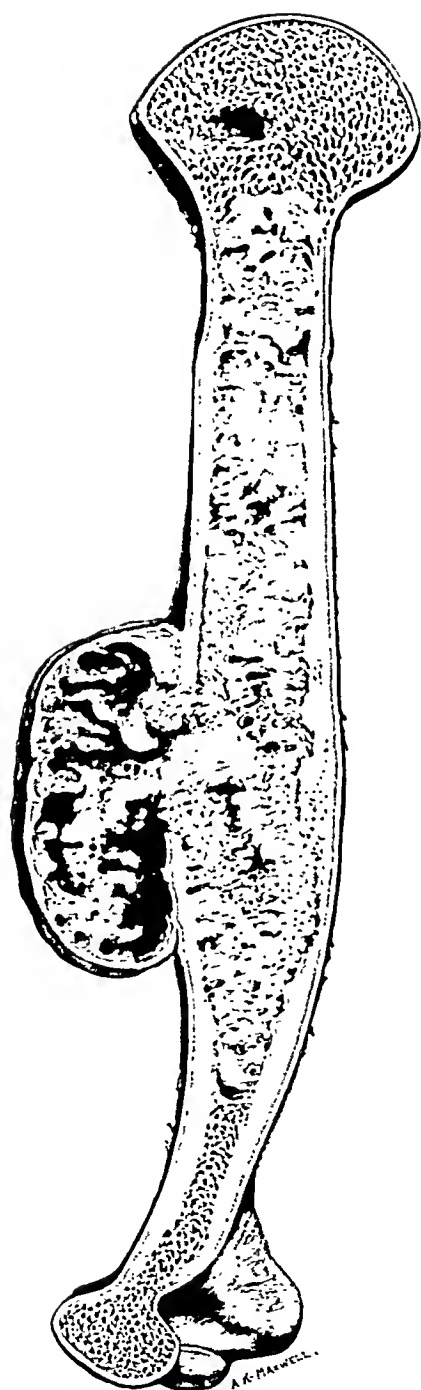
The greater part of the medullary canal is occupied by a chondroma which has slightly expanded the shaft. A short distance below the middle of the anterior surface the tumour has perforated the compact bone and has formed a hemispherical swelling beneath the periosteum.

The medullary portion of the tumour contains scattered foci of bone. The external portion is cystic from degeneration.

*Hunterian Museum, R.C.S. 1354.1*

MICROSCOPIC STRUCTURE.—Myxo-chondroma.

CLINICAL HISTORY.—The patient was a woman, aged 54, who had noticed a swelling of the arm for twelve months. The arm was successfully removed by amputation at the shoulder-joint.



## CHONDROMA OF HUMERUS.

A longitudinal section of an arm to the inner side of the humerus.

At the upper end of the specimen is the head of the humerus. From the lower end projects the stump of the forearm. The greater part of the cut surface is occupied by a chondroma, which measures 9 by 12 in. in diameter and surrounds the whole length of the bone except at the articular ends.

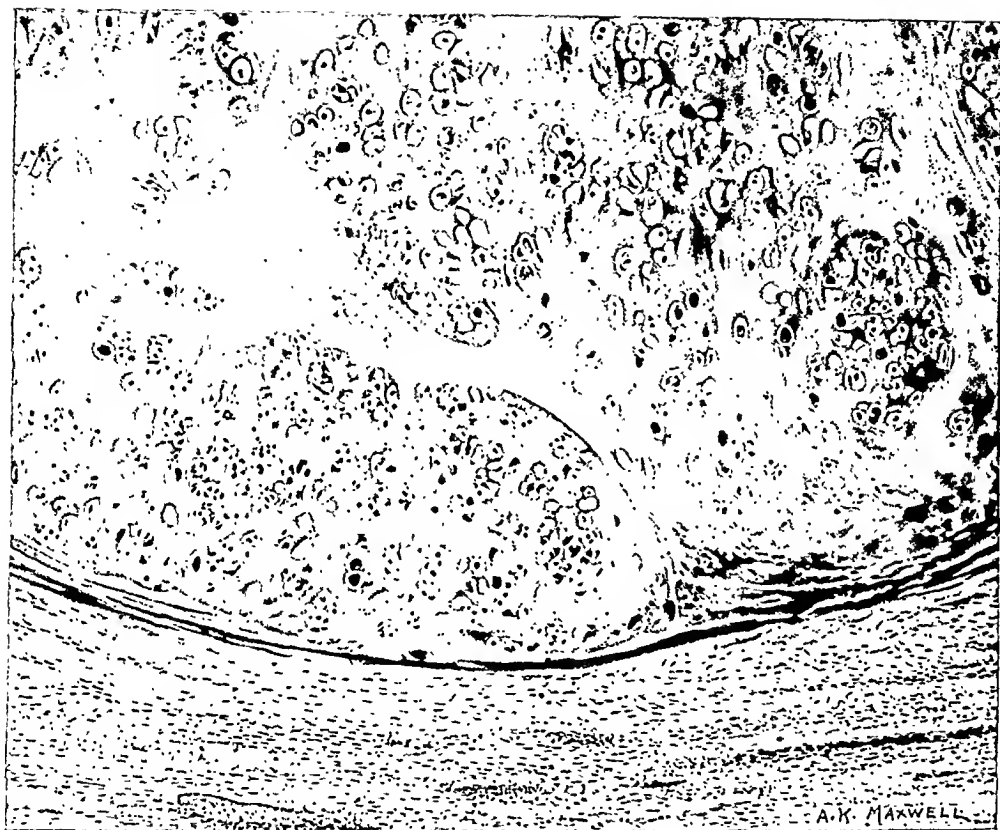
The surface of the chondroma is lobulated and surrounded by a fibrous capsule. Its substance is composed of translucent, hyaline cartilage in which are white areas of calcification and a few points of ossification. The cavities in the tumour were filled with a watery, mucinoid fluid, the product of degeneration in the cartilage.

*Hunterian Museum, R.C.S. 1342.2*

CLINICAL HISTORY.—The patient was a man, aged 46, who had noticed a painful swelling of the right arm for eight months. During the two months preceding amputation the growth had increased in size so rapidly and had been associated with so much emaciation as to suggest a malignant nature. There was no enlargement of lymphatic glands. Six years before there had been an injury to the right elbow. At the time it was not thought sufficiently severe to necessitate medical attention, but it had left the joint slightly stiff.

The limb was removed by a fore-quarter amputation in January, 1915. When last seen, in the summer of 1918, the man was quite well and had gained considerably in weight. There was no sign of recurrence, either locally or in distant parts.





**MICROSCOPIC STRUCTURE.**—The tumour is composed of hyaline cartilage, calcified in some parts, degenerate and liquefied in others. It is surrounded by a fibrous capsule.

## CANCELLOUS OSTEOMA OF GREAT TOE.



A longitudinal section of the terminal portion of a great toe.

A cancellous osteoma has grown from the dorsal aspect of the terminal phalanx and has displaced the nail. The free end of the tumour is nodular, and is capped by a thin layer of cartilage.

HUNTERIAN MUSEUM, R.C.S. 1363.1

## OSTEOMA OF FEMUR.

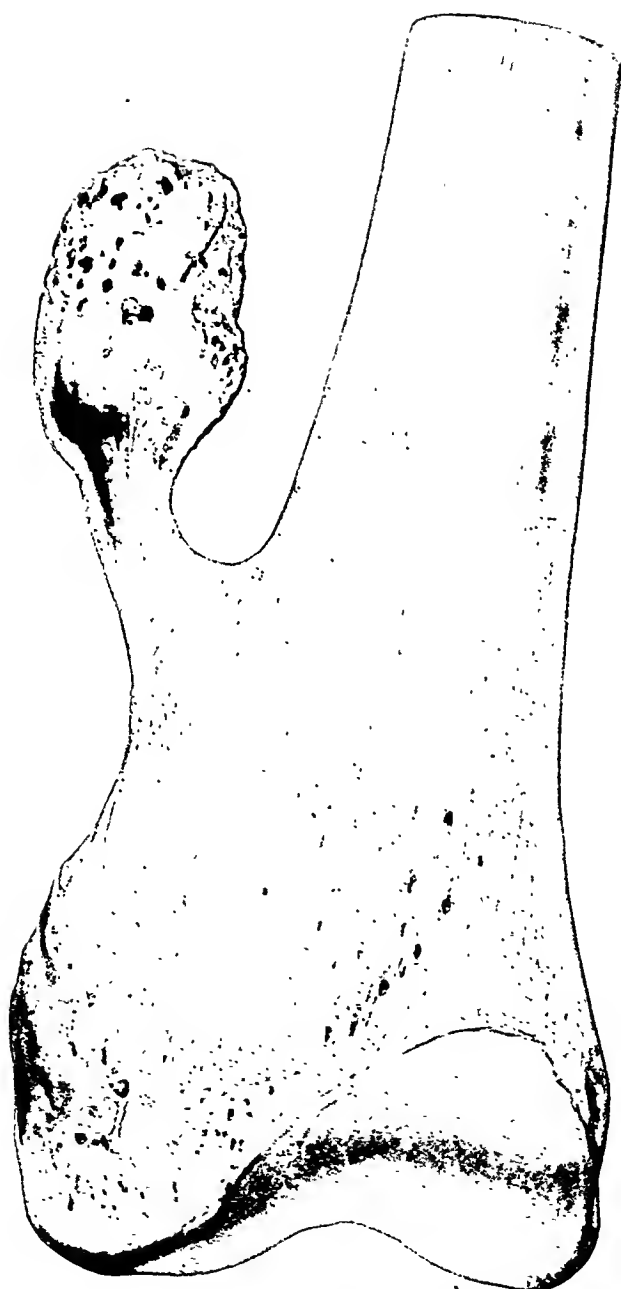
The lower end of a left femur.

A pedunculated, cancellous osteoma is attached to the inner border about one inch above the adductor tubercle. It did not lie within a muscle.

*Hunterian Museum, R.C.S. 1870.1*

CLINICAL HISTORY.—The patient was a man, aged 53, who had been accustomed to wrestling. He attributed the growth to a kick from a clog many years before death. There was no osteoma on the other femur.





A. K. MAXWELL.

### OSTEOMA OF SCAPULA.

The right scapula of a child.

A cancellous osteoma is attached by a broad base to the supraspinous fossa. Its surface is covered with cartilage, and shows the lobulation characteristic of an ossifying chondroma. *Hunterian Museum, R.C.S. 1872.1*

CLINICAL HISTORY.—The patient was a boy, aged 9 years, upon whose shoulder a painless swelling, which did not interfere with the movements of the arm, had been noticed for four weeks. There was no history of injury.

The scapula was successfully removed by operation.



HUNTERIAN MUSEUM, R.C.S. 1372.1

NO. 3—SUPPLEMENT

### OSTEOMA OF INNOMINATE BONE.

One-half of a cancellous osteoma, with a portion of the iliac crest to which it was attached.

The tumour arises by a narrow neck from the surface of the iliac fossa immediately below the epiphysis of the crest, and expands into a lobulated swelling coated with translucent cartilage. Beneath the layer of cartilage is an irregular, opaque, white zone of calcification. The rest of the osteoma is composed of cancellous bone continuous with that of the ilium.

*Museum of University College Hospital, 82.A.12.*

CLINICAL HISTORY.—The patient was a man, aged 18, who first noticed a painless lump on his left hip bone two years before. It was then about  $\frac{1}{4}$  in. in diameter, but had grown steadily since. There was no history of injury. On examination, a hard, nodular mass was felt in the left iliac fossa, attached to the inner side of the crest 1 in. behind the anterior superior spine. It was removed by operation.



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL. 82.A.12

## MULTIPLE OSTEOMATA OF FEMUR.

The posterior half of a right femur which has been divided longitudinally.

The upper and lower extremities and the adjoining portions of the shaft have been deformed by the growth of multiple cancellous osteomata. The overgrowth of bone has caused a diffuse enlargement of the lower and posterior portion of the neck above, and of the supracondylar region below. In addition, numerous sessile and pedunculated osteomata spring from the sites of muscular attachment along the gluteal ridge and around the adductor tubercle.

*Hunterian Museum, R.C.S. 1373.1*

The specimen was obtained from a graveyard.



### OSTEOMA OF SKULL.

The upper half of a horizontal section through the anterior portion of the skull.

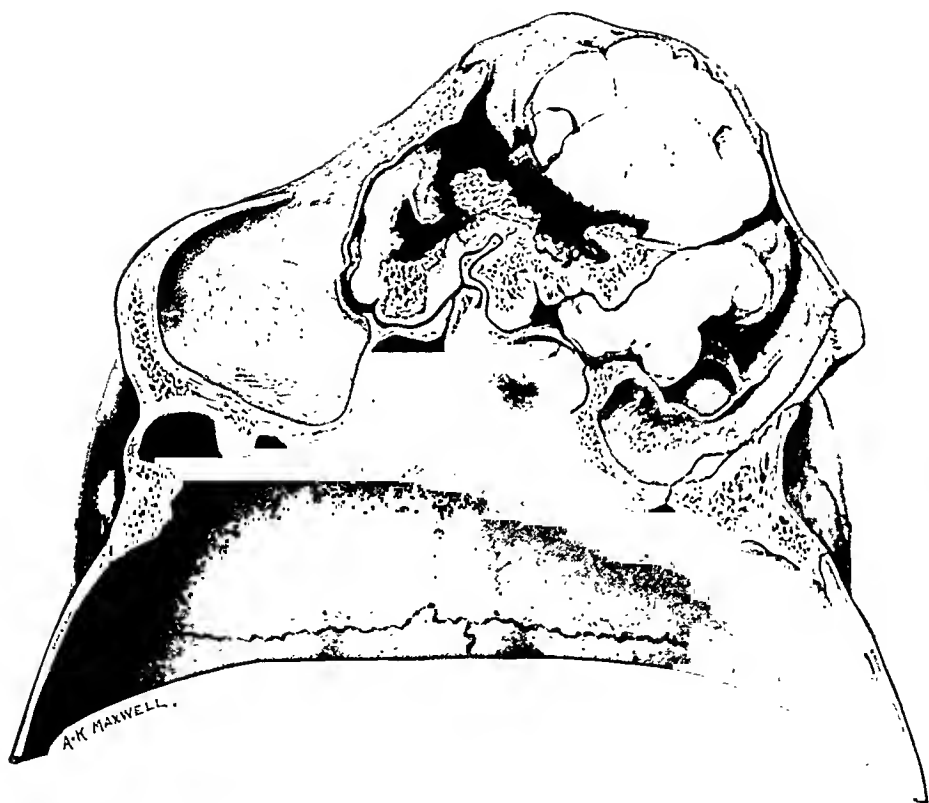
The frontal sinuses are expanded by a compact osteoma which has perforated their anterior walls and has invaded the cranial cavity and left orbit.

The tumour is irregularly nodular in outline, and, for the most part, has the appearance and density of ivory. The central portion is composed of cancellous bone.

*Hunterian Museum, R.C.S. 1390.1*

*[Hunterian Specimen]*





HUNTERIAN MUSEUM, R.C.S. 1390.1  
[HUNTERIAN SPECIMEN]

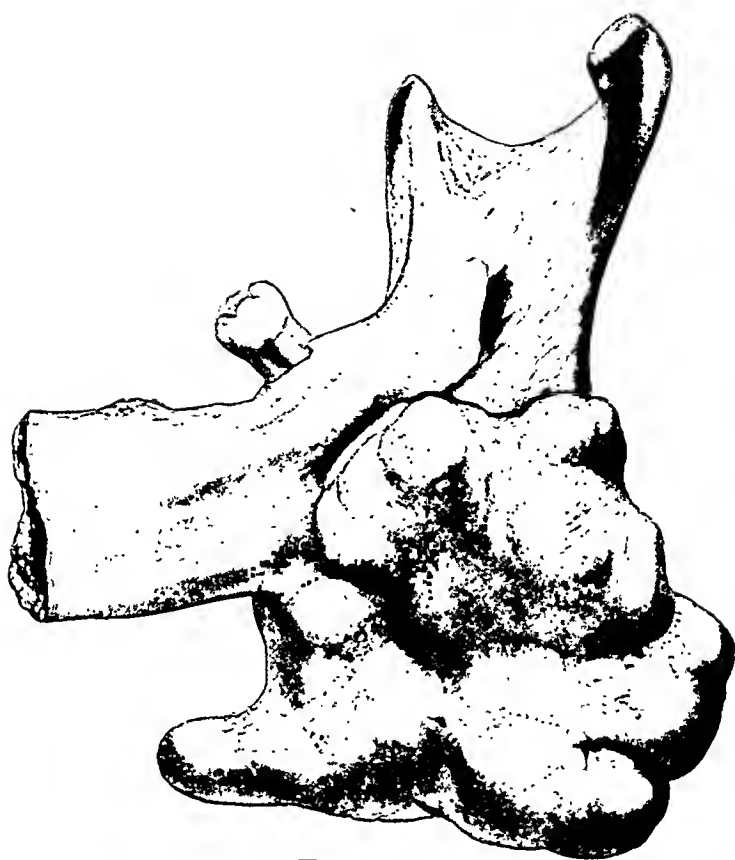
### COMPACT OSTEOMA OF LOWER JAW.

The greater part of the right half of a lower jaw.

A compact osteoma,  $2\frac{1}{2}$  in. in diameter, projects from the angle, to which it is attached over a small area above and in front. The angle of the jaw lies in a groove along the upper aspect of the tumour, and has been partially absorbed by its pressure.

The tumour has a lobulated surface, and an ivory-like density on section.

*Hunterian Museum, R.C.S. 1391.1.*



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### FIBROMA OF HUMERUS.

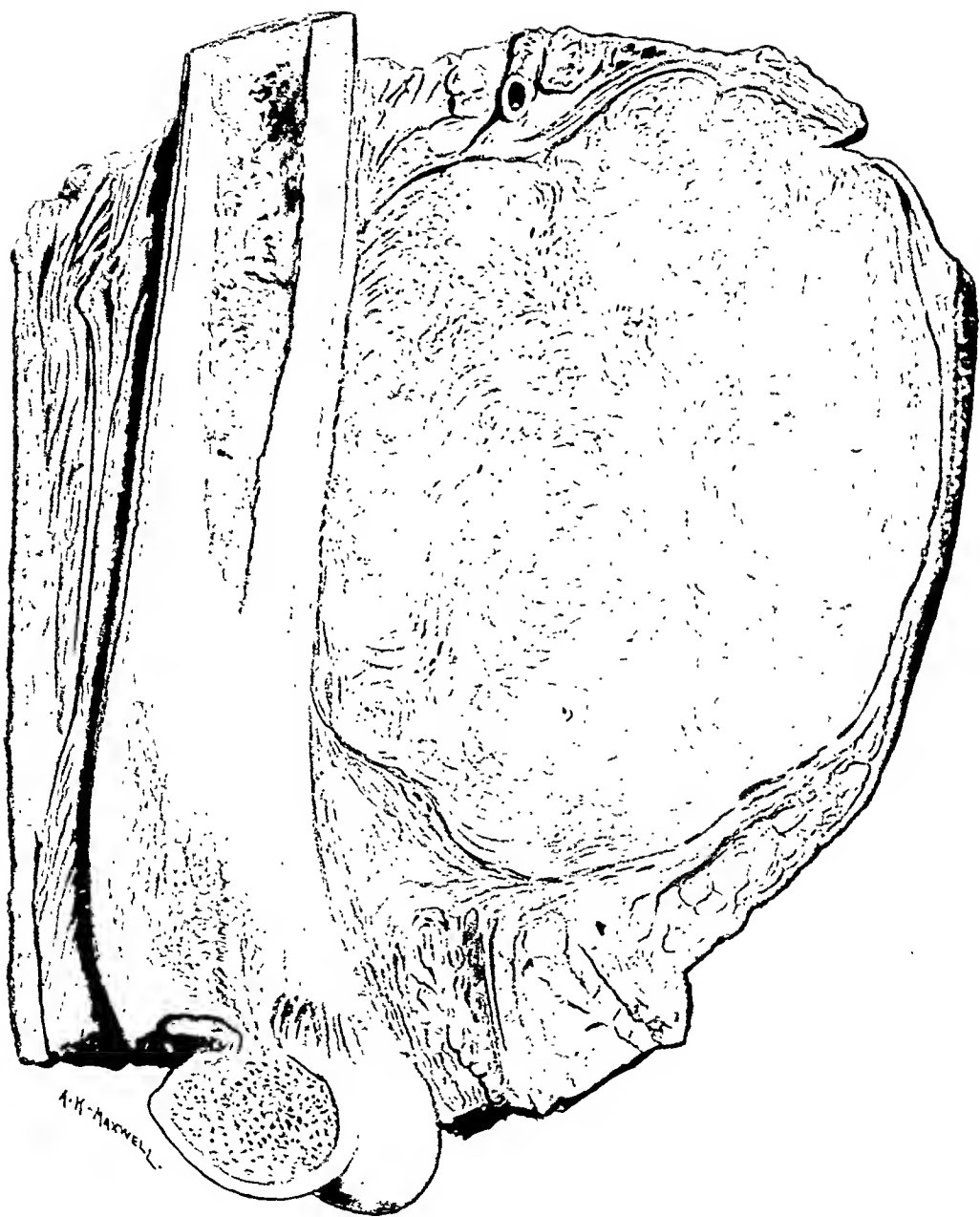
A sagittal section of the lower half of the arm.

An encapsuled fibrous tumour has grown from the front of the humerus and has displaced the soft parts. No periosteum can be distinguished along the attachment of the tumour, but the osseous tissue is not involved.

*Hunterian Museum R.C.S. 1296.1*

*[Hunterian Specimen]*

MICROSCOPIC STRUCTURE.—The tumour is composed of intersecting bundles of fibrous tissue. Small calcified areas are scattered throughout its substance.



HUNTERIAN MUSEUM, R.C.S. 1296.1  
[HUNTERIAN SPECIMEN]

## FIBROMA OF LOWER JAW.

The right half of a lower jaw.

The ramus is expanded by a fibrous tumour which has left unaltered only the incisor region and the condyle, and projects upwards as a lobulated mass above the bone.

*Museum of University College Hospital, 4.AF.8*

MICROSCOPIC STRUCTURE.—Delicate fibrous tissue.

CLINICAL HISTORY.—The patient was a man, aged 30, in whom a painless tumour had been growing for two and a half years. It extended chiefly upwards and backwards within the mouth, encroaching on the soft palate and rendering deglutition difficult. The specimen was successfully removed by operation.



## MYELOMA OF TIBIA.

A sagittal section of the lower end of a tibia, with the surrounding soft parts.

For 3 in. above the epiphysial cartilage the tibia has been expanded by a myeloma, and is composed of a thin shell of bone enclosing a central cavity. The interior is partly subdivided by incomplete septa, but is otherwise smooth, and is lined by a thin layer of growth, red in colour from extravasated blood, and sharply differentiated from the marrow of the shaft. At one point it has perforated the epiphysial cartilage. In the recent state the cavity contained a clear, watery fluid, tinged with blood.

The soft parts surrounding the expanded bone are displaced but not infiltrated.

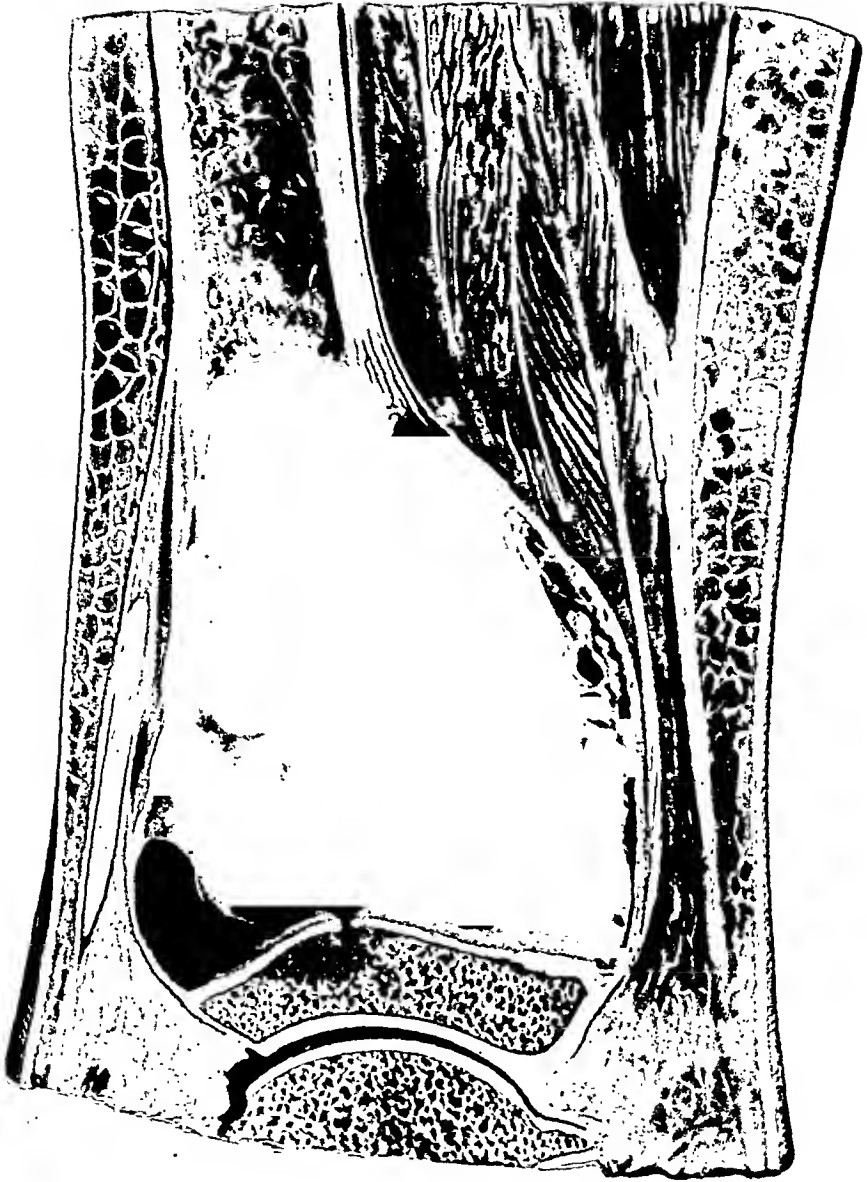
*Hunterian Museum, R.C.S. 1972.1*

MICROSCOPIC STRUCTURE.—The tumour consists of spindle-shaped cells among which are large numbers of multinucleated giant cells. It is highly vascular.

CLINICAL HISTORY.—The patient was a girl, aged 14, in whom an increasing swelling of the lower part of the left leg had been noticed for nine months. Pain was felt on walking.

(*H. A. Lediard, Proc. Roy. Soc. Med. (Clin. Sect.), 1911, 132.*)





A. H. MAXWELL

## MYELOMA OF FEMUR.

A coronal section of the lower part of a left femur.

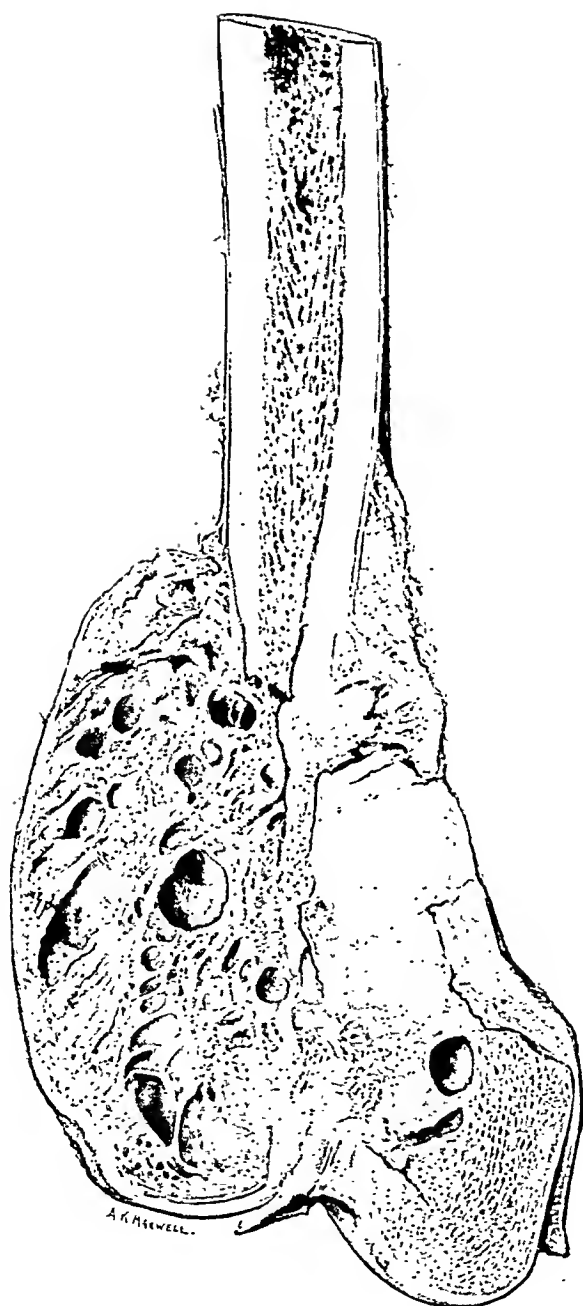
The lower extremity is deformed by a tumour which has expanded the external condyle. The growth is sharply differentiated from the shaft above, and from the cancellous tissue of the internal condyle, a considerable part of which is not invaded, on the inner side. Its external surface is bounded by periosteum. The articular cartilage of the external condyle is intact.

The cut surface of the tumour displays numerous cystic spaces, except over a structureless area above the internal condyle.

*Museum of St. Bartholomew's Hospital, 471.E*

**MICROSCOPIC STRUCTURE.**—The tumour consists of a groundwork of spindle cells, throughout which giant cells are thickly and uniformly scattered.

**CLINICAL HISTORY.**—The patient was a man, aged 54, who had noticed a swelling in the region of the left knee since an accident six months before. For the last three months of this period the swelling had increased rapidly in size. The limb was removed by amputation.



### MYELOMA OF RADIUS.

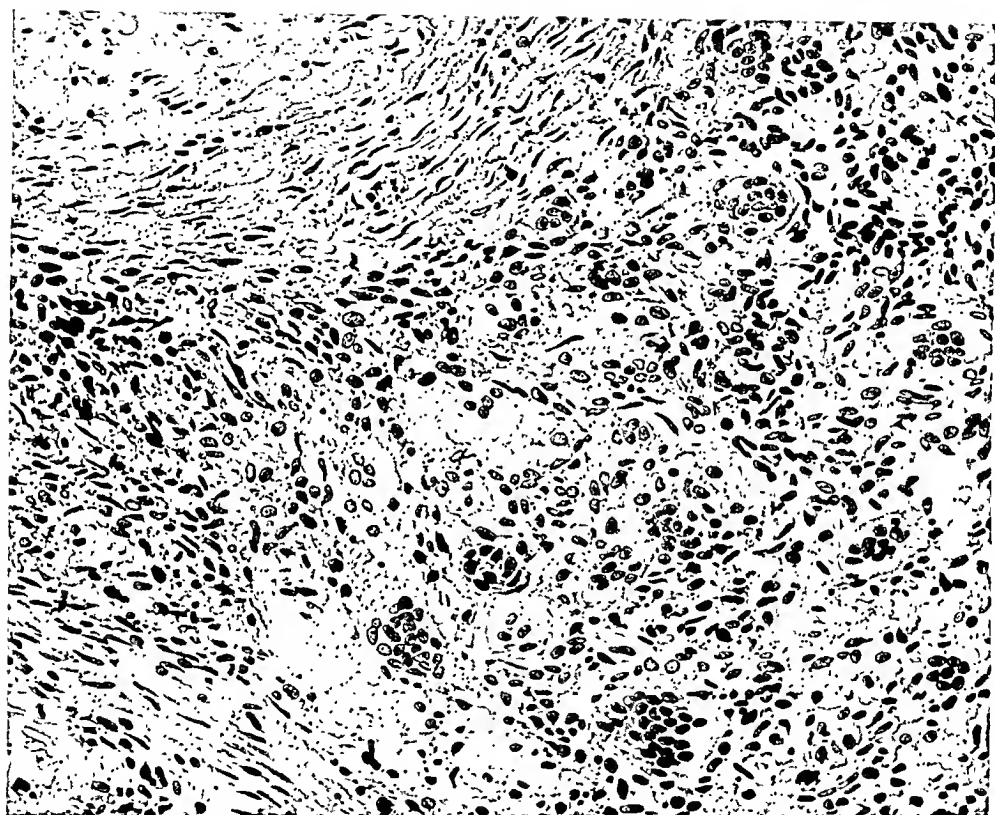
A sagittal section of the lower end of the forearm and the hand.

The lower end of the radius is expanded by a central tumour 10·2 cm. in diameter. The expanded portion is composed of a series of cysts of various sizes filled with a green, translucent material, probably mucoid in character. Some of the cysts contain blood. The tumour as a whole is surrounded by a fibrous capsule with which is incorporated an incomplete shell of newly-formed bone.

*Hunterian Museum, R.C.S. 1969.1*

**CLINICAL HISTORY.**—The patient was a woman, aged 37, who had sustained a Colles' fracture of the wrist four years before the date of amputation. The swelling had been present ever since the accident, and at the time of admission to hospital the skin over the tumour was of a dusky hue and had 'given way' at one point. No loss of flesh had been noticed. The patient was discharged well two months after operation in 1900. She has not been seen since.

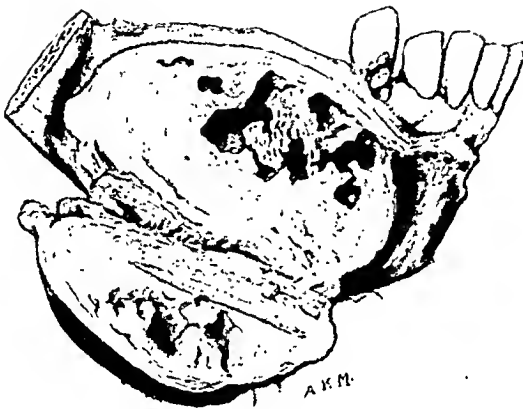




A. K. MAXWELL.

MICROSCOPIC STRUCTURE.—The solid tissue between the cysts near the surface of the growth is made up of spindle cells, among which are large numbers of multinucleated giant cells.

## MYELOMA OF MANDIBLE.



The symphysis and a portion of the horizontal ramus of a lower jaw.

Behind the incisor teeth the bone is expanded by a central tumour, from which a slice has been removed. The oval tumour is surrounded by a thin, bony shell, and presents a homogeneous appearance except in the areas where degeneration has occurred. The overlying mucous membrane is intact, but the bicuspid and molar teeth have disappeared.

MICROSCOPIC STRUCTURE.—Myeloma.

MUSEUM OF THE LONDON HOSPITAL 669

## MYELOMA OF JAW.

The greater part of the left half of a mandible.

From the condyle and coronoid process to the second molar tooth the jaw is expanded by a central tumour. The colour of this tumour is dark red, with small yellowish-brown areas. Its consistence is so soft as almost to be gelatinous.

*Museum of the Middlesex Hospital, C.183*

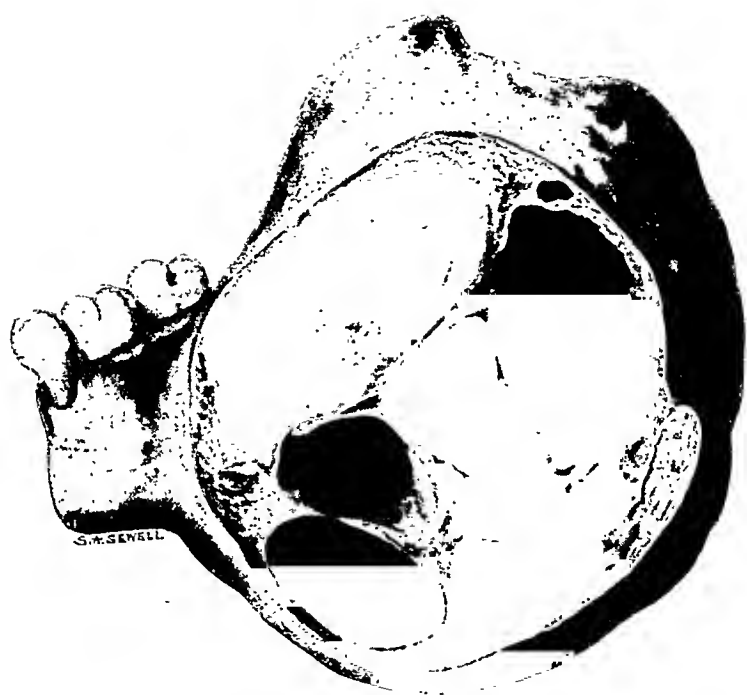
**MICROSCOPIC STRUCTURE.**—The growth consists in part of firm, fibrous tissue, in part of a delicate stroma containing spindle cells and multinuclear cells of myeloid type, with an infiltration by polymorphonuclear leucocytes.

**CLINICAL HISTORY.**—The patient was a boy, aged 15, below whose left ear a small and slightly painful swelling had first been noticed seven months before admission to hospital. The lump increased gradually in size and became painless as it grew. On examination, a hard swelling, fixed to the jaw, occupied the left parotid region. The neighbouring lymphatic glands were not enlarged.

X-ray examination showed disorganization of the left ramus of the mandible.

The tumour, together with the greater part of the left half of the lower jaw, was removed by operation.





### III. ODONTOMES.

“AN odontome is a tumour composed of dental tissues in varying proportions and different degrees of development, arising from teeth-germs, or from teeth still in the process of growth.”

The definition is Bland-Sutton's, and it is his classification, as modified by the Committee of the British Dental Association, which is here adopted. This classification includes, in addition to obvious tumour-formations, the dental cyst, the origin of which is, in many cases, closely connected with infection around the apex of a tooth. It includes also certain aberrations in the development of teeth—geminated and gestant composite odontomes, and enamel nodules. In the group of composite odontomes many are of small size, remain closely connected with the teeth from which they originate, and are seldom included within the experience of the general surgeon.

The odontomes, as a whole, are benign tumours of slow growth which expand the part of the jaw in which they lie. They seldom cause pain unless secondarily infected. They are classified in three main groups, according to the part of the tooth-germ from which they arise. The epithelial odontomes are formed through an abnormal development of the dental epithelium alone, the composite odontomes arise from the dental epithelium and other dental tissues, and the connective-tissue odontomes spring from the connective tissues surrounding the tooth-germ.

The classification adopted is as follows:—

1. *Epithelial odontomes*: (a) Multilocular cyst; (b) Dentigerous cyst; (c) Dental cyst.
2. *Composite odontomes*.
3. *Connective-tissue odontomes*: (a) Fibrous odontome; (b) Cementome.

#### 1. Epithelial Odontomes.—

*a. MULTILOCULAR CYST.*—The multilocular cyst is an encapsuled tumour situated centrally within the jaw, which is expanded so as to form a thin shell around it. The majority of specimens preserved in museums have grown for many years and have reached a large size before removal. The external surface of the tumour is generally smooth, but a slight lobulation may indicate its multilocular structure.

On section, it is made up of a number of cysts of varying size, separated by septa of varying thickness which may contain bone. The proportion of solid tissue to cyst differs considerably in different specimens, and the relative amount of solid tumour substance is often increased by the presence of papillary growths within the cysts. The growing portion of the tumour has a reddish tinge. The cysts, when free from intracystic growths, have a smooth lining and contain clear mucoid or brown serous fluid. The larger ones may communicate with each other by absorption of the intervening partitions.

Microscopic examination shows that the solid tissue between the cysts is composed of branching columns of epithelial cells embedded in a connective-tissue stroma. The peripheral cells of the columns are usually cylindrical

in shape, the central cells are small and rounded or oval. The cysts arise through degeneration of these cells, which may assume a stellate form in the process. As a cyst increases in size its lining epithelium becomes flattened and may even be difficult to identify. Occasionally a section shows the cell-columns of the tumour continuous with the epithelium of the alveolar mucous membrane, when it recalls the appearance of a basal-celled carcinoma of the skin. In this connection it may be observed that a pigmented epithelial odontome has been described.

The multilocular cyst is far more common in the mandible than in the maxilla, and twice as common in women as in men. It occurs at any age, but in about half the cases has first been noticed between 20 and 35. As the tumour increases in size it pushes out the adjacent teeth, and may become infected from the mouth, though this complication is not so common as in the case of the solid odontomes.

**b. DENTIGEROUS CYST (Follicular Odontome).—**The dentigerous cyst is a unilocular cyst with an epithelial lining and a fibrous wall, to which is attached an unerupted tooth. The surrounding jaw is expanded.

The fibrous wall varies in thickness and may contain calcareous particles. It rarely becomes calcified throughout. The epithelial lining presents an intriguing variety. It may be a thin, stratified layer recalling that of the dental cyst, or a thick layer of columnar and cubical cells degenerating towards the lumen. Papillary processes may project from its surface into the cavity, and in some cases it repeats the histological structure of the multilocular cyst. The fluid contents are viscid, and may hold cholesterol crystals and epithelial debris in suspension. Suppuration sometimes occurs, but is not common. The associated tooth usually has an ill-developed root embedded in the wall and a well-formed crown projecting into the cavity of the cyst, but may lie free within the lumen. It is almost always one of the permanent set, most commonly the canine, but in a few instances dentigerous cysts have been described in connection with deciduous or supernumerary teeth.

The dentigerous cyst is commonly single, occasionally multiple, and occurs with equal frequency in maxilla and mandible. It is found in both sexes at all ages, though most often in childhood and early adult life.

**c. DENTAL CYST.—**The dental cyst is a unilocular cyst lined by epithelium and associated with a normally-erupted tooth of the permanent set. In rare cases it is found in connection with a deciduous tooth. It begins round the root of a dead tooth, usually a molar, by proliferation of the remnants of the dental epithelium, which are stimulated to growth by the products of infection. The mass of epithelium undergoes degenerative liquefaction in the centre, and around its periphery acquires a fibrous wall through inflammatory reaction in the surrounding tissues. Small sacs, formed in this way, are often found adhering to the roots of teeth extracted on account of infection, and it is probable that dental cysts have a similar origin, and differ only in the persistence of their growth.

The epithelium lining a dental cyst is usually stratified, but the shape and arrangement of the cells are subject to considerable variation in agreement with the potentialities of the dental epithelium from which it is

derived. The fluid in the cyst is serous, and frequently contains cholesterin crystals. Suppuration occasionally occurs.

Dental cysts are more common in the maxilla than in the mandible, and frequently project into the antrum. They form smooth, more or less spherical swellings which expand the jaw, and sooner or later cause absorption of some part of their bony shell. Through the soft spot thus formed the fluid nature of the swelling can be recognized.

## 2. Composite Odontomes.—

*Complex Composite Odontomes.*—The commonest variety of composite odontome is an irregular conglomerate of enamel, dentine, and cementum, which forms a nodular, calcified tumour within the jaw. To the naked eye it may in no way resemble a tooth, but a dental origin is often suggested by the presence of fragments of white or pigmented enamel scattered irregularly on its surface or throughout its substance. In the centre of the tumour the tissues are confused. In the peripheral part enamel and dentine are often arranged with sufficient regularity to appear as alternating layers radiating towards the surface. Holes and slits lined by enamel occur here and there. The pulp-cavity is represented by multiple, irregular canals. Complex composite odontomes are slowly-growing tumours which expand the jaw, most often in the molar region. Apart from the swelling and a moderate discomfort, the chief symptoms are those dependent on infection. The tumours affect either jaw in either sex, and they first appear in youth or early adult life.

*Compound Composite Odontomes.*—In this, a less common variety, recognizable denticles are formed within a mass of vascular tissue, variably calcified and sometimes cystic. The denticles may be few in number, or some hundreds may be present. The tumour is usually enclosed in a thin, calcified capsule, but its friable nature does not lend itself to removal in an intact state.

In the group of composite odontomes are also included certain pathological formations which remain small in size and closely connected with the tooth from which they spring:—

*Geminated Composite Odontomes.*—These are composed of malformed teeth fused together. The fusion may involve any portion of the individual denticles, and the pulp-cavities may be combined or separate. The incisor region is the usual site.

*Gestant Composite Odontomes.*—In this variety a denticle is enclosed within a tooth.

*Enamel Nodules.*—These project from the dentine of a tooth, and are either separate from or connected by a narrow neck with the normal enamel. Each consists of a boss of dentine capped by enamel. Beneath the dentinal projection there may be a prolongation of the pulp-cavity. Enamel nodules are usually found between the roots of upper molar teeth.

*Dilated Composite Odontomes.*—Small tumours which bear a recognizable resemblance to teeth, from the normal shape of which they depart by a local increase in diameter and by the inclusion of a central cavity. They have the microscopical structure of a complex composite odontome. Various subdivisions are described, among the most important being the radicular odontome.

### 3. Connective-tissue Odontomes.—

*a.* FIBROUS ODONTOME.—The fibrous odontome is an encapsuled fibrous tumour derived from the connective-tissue sheath of the dental sac. Within it, or attached to it, is a tooth. It is rare in man, and appears to be associated with rickets.

*b.* CEMENTOME.—A cementome is a tumour composed of cementum, and, in its most characteristic form, is connected with the roots of a mandibular molar. The related tooth is not deformed. It is rare in man, and, like the fibrous odontome, may be associated with rickets. The cementome, on section, is not laminated, and in this particular presents a structural difference from the laminated cementum which is often deposited upon roots in consequence of inflammatory irritation.

In connection with the connective-tissue odontomes, it may be pointed out that osseous odontomes have been described in relation both to the crowns and to the roots of teeth. The origin of such tumours is ascribed to the layer of bone which immediately surrounds the follicle.

## MULTILOCULAR CYST.

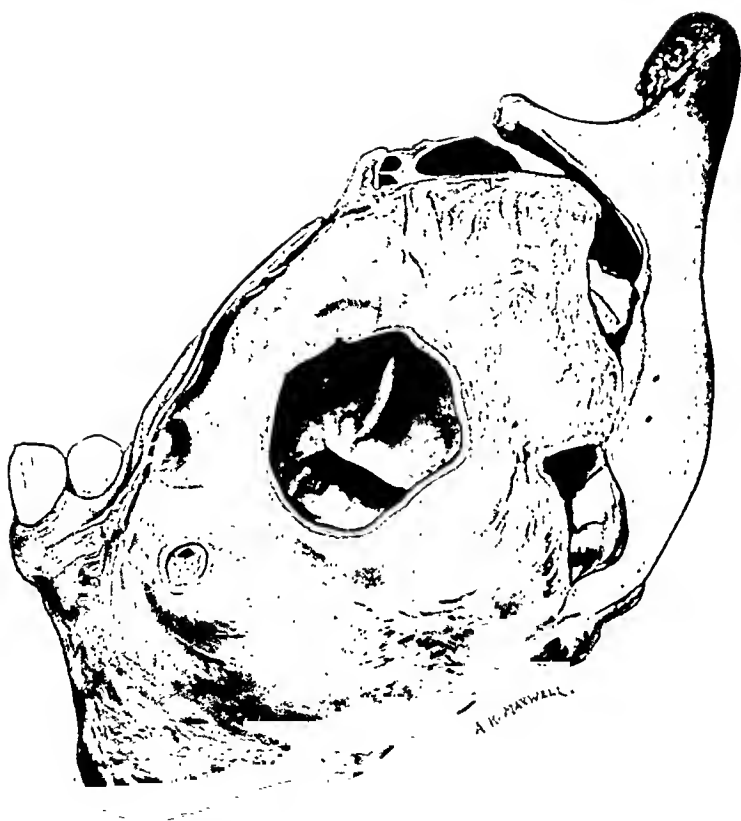
The left side of a lower jaw, from the condyle to the canine tooth.

The body and ascending ramus are expanded by an oval, multilocular, cystic tumour with a lobulated surface. Its outer bony wall is very thin in parts, and is no longer continuous with the posterior portion of the ramus. There is an ulcerated opening in the gum covering the tumour, corresponding with the socket of the first molar tooth. *Hunterian Museum, R.C.S. 1406.1*  
[From the Museum of Robert Liston]

**MICROSCOPIC STRUCTURE.**—The tumour is composed of columns of small, round, epithelial cells in a matrix of well-developed fibrous tissue. A few of the alveoli are lined by a layer of cylindrical epithelium enclosing a delicate network of stellate cells. Around the sinus into the mouth the epithelium of the tumour is continuous with that of the mucous membrane covering the jaw.

**CLINICAL HISTORY.**—The patient was a middle-aged man in whom the disease had existed for several years. The cyst, which was first formed in the situation of the last two molars, had been regarded as a simple cavity in the bone containing fluid, and setons had been passed through it with seeming benefit, but the swelling returned and increased rapidly. The parts shown were removed by operation, which was permanently successful.

(*Eve, Brit. Med. Jour.*, 1883, i, 1.)



*Natural size*

## MULTILOCULAR CYST.

A tumour removed from the lower jaw.

The surface is lobulated and covered by a capsule. On section, it is made up of cysts of varying size with intervening septa of varying thickness. The lining of the cysts is smooth for the most part, but a few papilliferous outgrowths are present. The larger cysts communicate with one another, and some contain small quantities of altered blood.

*Museum of the Middlesex Hospital, C.187*

**MICROSCOPIC STRUCTURE.**—The tumour is composed of columns of epithelial cells lying in a connective-tissue stroma. The outermost cells of the columns are elongated and bear a close resemblance to the basal cells of the skin. The inner cells are more squamous in character. The cysts contain papillary ingrowths of epithelium.





*Natural size*

## EPITHELIAL ODONTOME.

(MULTILOCULAR CYST.)

A portion of the left maxilla.

The bone is distended by an encapsuled tumour which extends from the palate to the roof of the antrum. On section, the growth is mostly solid, with areas of softening.

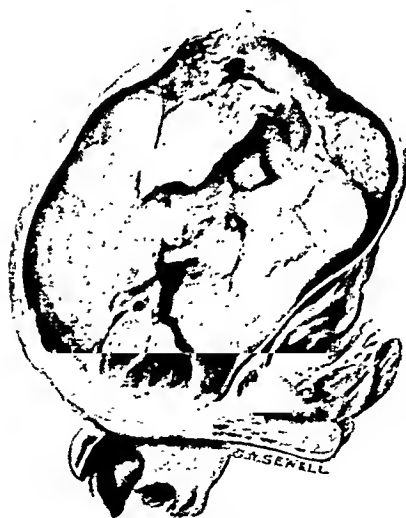
*Museum of St. Bartholomew's Hospital, 538.B*



× 45

**MICROSCOPIC STRUCTURE.**—The tumour is composed of columns and masses of epithelium lying in a connective-tissue stroma. Its structure is characteristic of multilocular cystic disease.

**CLINICAL HISTORY.**—The patient was a man, aged 60, from whom two bicuspid teeth had been removed on account of toothache six months before admission to hospital. The pain was not relieved, and shortly afterwards the swelling appeared, with an intermittent discharge from the nose.



*Natural size*

## DENTIGEROUS CYST.

One half of a dentigerous cyst which was removed from the lower jaw.

The wall is formed by a thick layer of fibrous tissue with granulation tissue on its inner surface. No epithelial lining can be demonstrated. A well-formed bicuspid tooth lies within the cavity, to the wall of which it is attached by fibrous tissue. The cyst was filled with albuminous fluid.

*Hunterian Museum, R.C.S. 4185.1*

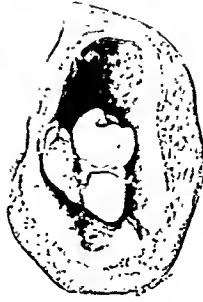
CLINICAL HISTORY.—The patient was a boy, aged 14. Five months before the operation he received a blow on the right side of the lower jaw. The gum bled profusely, and from that time the jaw gradually swelled. The tumour was considered at first to be an exostosis. The patient recovered.

*(Lancet, 1850, i, 756.)*

## DENTIGEROUS CYST.

A right superior maxilla.

The antrum is partly filled by a calcified cyst into which opens the socket of the second premolar tooth. The separate tooth shown at the back of the specimen was described by Christopher Heath as a supernumerary tooth loose within the cavity of the cyst. *Hunterian Museum, R.C.S. 4180.2*



*Natural size*

HUNTERIAN MUSEUM R.C.S. 4185.1



*Natural size*

HUNTERIAN MUSEUM, R.C.S. 4180.2

## DENTIGEROUS CYST.



*Natural size*

A dentigerous cyst containing a tooth.

The cyst is spherical in shape, with a fibrous wall. A portion of the latter has been removed, and through the opening can be seen a smooth lining and the well-formed crown of a bicuspid tooth projecting into the cavity. In the recent state the cyst contained mucopus.

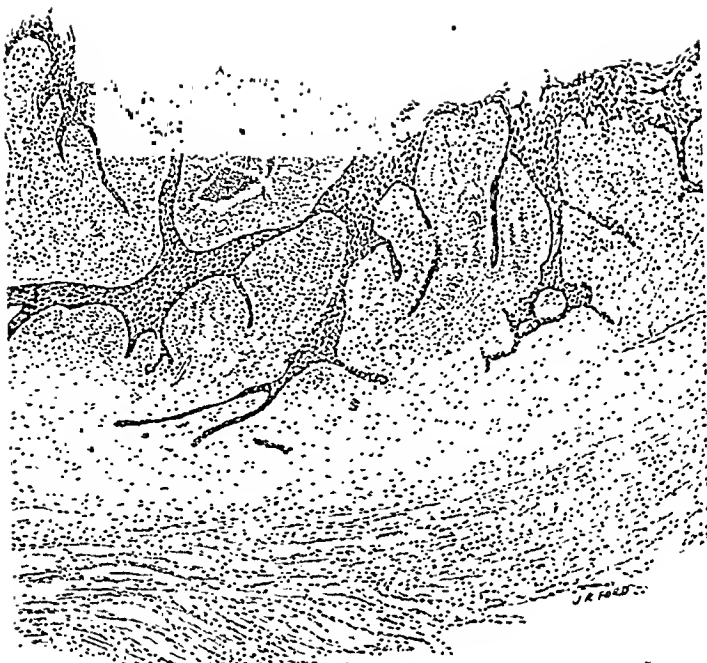
*Museum of University College Hospital, 11.AF.1*

**MICROSCOPIC STRUCTURE.**—The wall of the cyst is composed of fibrous tissue infiltrated by round cells. It is lined by a discontinuous layer of stratified epithelium, from the deep surface of which columns of epithelial cells project into the subjacent fibrous tissue.

**CLINICAL HISTORY.**—The patient was a boy, aged 6 years, who had a swelling of the right lower jaw for eight weeks. A tooth had been extracted from the neighbourhood of the swelling one year previously. The incisors and canine, but no other teeth, had erupted from the right lower jaw, in which a hard swelling, about the size of a cherry, could be felt arising gradually from the outer aspect. The inner side of the jaw was slightly expanded. The swelling was everywhere of bony hardness except in the highest part of the gum, where fluctuation was present. In the gum at the back of the swelling was a sinus through which a probe passed to an unerupted tooth.

X-ray examination showed the jaw expanded by a smooth-walled cavity which contained a tooth.

The cyst was enucleated through the mouth after removal of the outer portion of its bony shell.



x 15

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL 11.AF.1



## DENTAL CYST.

A mandibular molar with a dental cyst attached to the end of one of the roots.

*Hunterian Museum, R.C.S., Odontological Collection, D.158.12*

## DENTAL CYST.

The right half of a section through the anterior portion of a skull, seen from the cut surface.

The bone has been divided along a line which passes from the centre of the posterior border of the hard palate to the gap between the right central and lateral incisors.

The anterior part of the maxilla is distended by a cavity in which the root of the lateral incisor is exposed. The other half of the specimen presents a similar appearance. There is no evidence of infection in the bone surrounding the cavity or in the related teeth.

*Hunterian Museum, R.C.S. D.159.1*

There is no history to the specimen.





*Natural size*

HUNTERIAN MUSEUM, R.C.S., CRONTOLOGICAL COLLECTION, D.159.12



*Natural size*

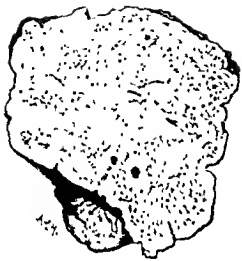
HUNTERIAN MUSEUM, R.C.S., D.159.1

## COMPLEX COMPOSITE ODONTOME.

One half of an odontome which was removed from the lower jaw.

The shape of the tumour is irregularly oval, and its surface is covered with projecting nodules, some of which are capped by enamel. The section shows that it is made up chiefly of folds of dentine which surround flattened pulp-cavities. There is also a small quantity of enamel which, besides covering some of the excrescences on the surface, dips down into the tumour to follow the convolutions of the dentine.

*Hunterian Museum, R.C.S. 1404.1*



*Natural size*

**CLINICAL HISTORY.**—The patient was a woman, aged 18, who had been rickety in childhood. The first teeth came late and decayed rapidly. Nothing abnormal was noticed about the second dentition. For eight months before removal of the tumour she had felt pain and uneasiness, for which several teeth were extracted. Ultimately the odontome became exposed, but until it was removed it was thought to be a sequestrum.

*(Tomes, Trans. Clin. Soc., 1882, xv, 10.)*



*Natural size*

## ENAMEL NODULE.

An enamel nodule between the roots of a molar tooth.

*Hunterian Museum, R.C.S., Odontological Collection, C.83.1*

## DILATED COMPOSITE ODONTOME.

(RADICULAR TYPE.)

A molar tooth, on which, attached to one side of its root, and diverging at a right angle from it, there is a growth which, originally, was twice the size of the tooth.

A section has been made, and shows an outer layer of cementum, within which is an incomplete ring of dentine. The calcified centre contains numerous lacunae and scattered patches of dentine.

*Hunterian Museum, R.C.S., Odontological Collection, H.106*  
[*Hunterian Specimen*]

(*Salter, Guy's Hosp. Rep.*, 1869, xiv, 463, and 1876, xxi, 213.)



Natural size

## DILATED COMPOSITE ODONTOME.

(RADICULAR TYPE.)

A radicular odontome in connection with a maxillary molar.

The tumour is divided by two transverse grooves into three lobes of unequal size. Its surface is irregular. Two apertures lead into a large cavity in its centre.

*Hunterian Museum, R.C.S., Odontological Collection, D.166.1*

CLINICAL HISTORY.—The tooth was removed from a man, aged 41, who for some years had suffered severe pain in his upper jaw. A sinus passed out from the tumour to open on the cheek.

(*Tomes, Trans. Odontol. Soc.*, 1863, iii, 335, and iv, 81.)



Natural size

## COMPOUND COMPOSITE ODONTOME.

(RADICULAR TYPE.)



Natural size

A central incisor from the right maxilla, with an odontome growing from the internal aspect of its root.

*Hunterian Museum, R.C.S., Odontological Collection, D.170.1*

MICROSCOPIC STRUCTURE.—The tumour is composed of enamel and cement, with a few dentinal tubules.

(Colyer, *Trans. Odontol. Soc.*, 1906, xxxviii, 245.)



Natural size

## OSSEOUS ODONTOME.

An odontome covering the crown of a first mandibular molar.

Half of the odontome has been removed to show its relation to the tooth. The tumour is composed of cancellous bone surrounded by a fibrous capsule.

*Hunterian Museum, R.C.S., Odontological Collection, D.167.3*

## CEMENTOME OF MANDIBLE.



*Natural size*

HUNTERIAN MUSEUM, R.C.S. 1402.1

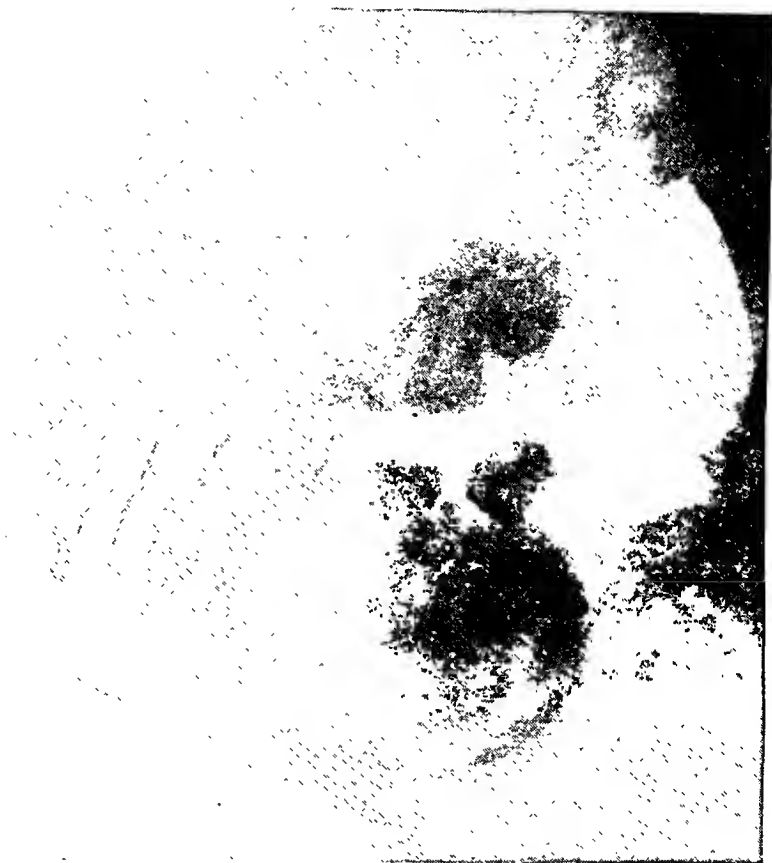
A cementome surrounding the roots of a second left mandibular molar.

The tumour is roughly spherical in shape, and is made up of bony trabeculae which radiate outwards from the tooth. In the recent state its interstices were filled with ossifying connective tissue.

**MICROSCOPIC STRUCTURE.**—Bone of open texture in a fibrous matrix.

**CLINICAL HISTORY.**—The patient was a boy, aged 15 years, in whom a swelling of the left side of the lower jaw had been noticed for a year. The tumour was opaque to X-rays. It was turned out of its bed, after removal of the shell of bone on its outer side, through an incision along the lower border of the mandible.

(*Bland-Sutton, Trans. Odontol. Soc.*, 1906, xxxviii, 213.)



The skiagram shows the dense shadow of a calcified tumour surrounding the roots of the second molar and expanding the jaw.

*Hunterian Museum, R.C.S., 1402.1*

